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Challenges and outcomes of management of anterior abdominal wall defects in a Nigerian tertiary hospital

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ABSTRACT

Background: Abdominal wall defect presents a great challenge when it is large, ruptured, or associated with other anomalies. **Objective**: To review the challenges and outcome of management of anterior abdominal wall defects (AAWD). **Materials and Methods**: A retrospective review of omphalocele and gastroschisis managed over 8 years at our institution. **Results**: Omphalocele (n=49) and gastroschisis (n=7) constituted 2.4% of total admission. The median age was 23.5 hours, with male-female ratio of 1:1.1. Term infants were 91.7% and more than 75% weighed above 2.5 kg. The mean maternal age was 28.5±5.87 years and mean parity was 3.1±2.0, with *P* values of 0.318 and 0.768, respectively. More than 92.9% of infants were out-born, 46 pregnancies (82.1%) were booked, and 51 (91.1%) had at least one ultrasound scan, but only 1 (1.8%) was diagnosed with gastroschisis. Ruptured omphalocele were 11 (6 major, 5 minor) in number, 3 of which presented with enterocutaneous fistula, and 3 (6.1%) were syndromic omphalocele. Positive blood culture confirmed septicaemia in 21 cases (37.5%). Surgical repair was done in 35 cases (62.5%), 44.6% as emergency, and 17.9% as elective. Non-operative management was done in 21 patients (37.5%) and 5 (8.9%) were discharged against medical advice. Median length of hospital stay was 10 days (mean, 15.98±14.38). Postoperative complication rate was 32.1% and overall mortality was 30.4%, with the highest case fatality among gastroschisis (57.1%) and omphalocele major (32.1%). **Conclusions:** There were large numbers of out-born infants due to poor prenatal diagnosis in spite of high instances of antenatal ultrasound scan. Many patients presented with complications that resulted in poor outcome.

Key words: Abdominal wall defects, challenges, gastroschisis, omphaloceles, outcomes

INTRODUCTION

Abdominal wall defects comprising both gastroschisis and omphalocele remain a source of significant morbidity and mortality, despite the advances in neonatal and paediatric surgical care.[1-3] Over the past few decades, survival has improved in developed countries, because of increased understanding of the in utero and antepartum diagnosis, especially with the availability of mechanical ventilation, parenteral nutrition, and various surgical repair techniques.[1,4,5] However, controversy remains regarding aetiology, anatomy and embryology, the role of prenatal diagnosis and mode of delivery, as well as initial management.[6]

In developing countries, anterior abdominal wall (AAW) anomalies still pose a challenge of diagnosis and management among healthcare givers, especially in complicated cases.[2,7] This may arise due to uncommon presentation of such cases in the peripheral health centres where a majority of children are delivered. This study highlights the challenges and outcomes of management of anterior abdominal wall defects at a tertiary health centre in the North-Central geopolitical zone of Nigeria.

MATERIALS AND METHODS

We retrospectively reviewed cases of omphalocele and gastroschisis excluding seven cases of omphalocele associated with bladder extrophy managed by the paediatric surgery unit between January 2002 and December 2009. The biodata, clinical condition at presentation, interventions, and outcome were documented and analysed using SPSS version 16 software.

RESULTS

During the study period, a total of 2,381 children were treated by paediatric surgery unit PSU, of which 56 (2.4%) had omphalocele (n=49) and gastroschisis

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(n=7). The age at presentation was in the range 2-169 hours (median, 23.5 hours). The male-female ratio was 1:1.1 [omphalocele 1:1.2, gastroschisis 2.5:1]. Term infants were 91.7%, more than 75% of which weighed over 2.5 kg [Table 1]. Maternal age range was 17-43 years (mean, 28.5±5.9) and parity range was 1-8 children (mean±SD, 3.1±2.0). Maternal age and parity had no statistical significance in either of the anterior abdominal wall defects (AAWD) (P=0.318 and P=0.768, respectively).

A majority of patients were delivered in private clinics (42%), primary health centres (21%), and other places [Table 2]. There were 46 (82.1%) antenatal booked mothers and 51 (91.1%) had at least one ultrasound scan during pregnancy. However, only 1 (1.8%) had a prenatal ultrasound diagnosis of gastroschisis.

There were 11 ruptured omphaloceles (6 major, 5 minor), 3 of which had enterocutaneous fistula through the sac following dressing at home [Figure 1]. Omphalocele minor were 15 (30.6%), while omphalocele major were 34 (69.4%), with a mean size of the defect 7.5×6.3 cm. There were 3 cases (6.1%) of syndromic omphalocele, 2 of which were Beckwith-Wiedemann [Figure 2] and 1 epigastric omphalocele. All patients were at risk of sepsis at presentation, with positive blood culture of organisms in 21 (37.5%) and other presenting complications [Table 3].

All patients were resuscitated and treated as shown in Table 4. Surgical repair was done in 35 cases (62.5%)—25 (44.6%) as emergency ≤48 hours (omphalocele [11 minor (5 ruptured), 7 major (6 ruptured)], 7 gastroschisis) and 10 [(17.9%); 2 minor, 8 major] as elective repair. Non-operative management of the defect by the use of antibiotic and sac dressing was done in 19 patients [(33.9%); 2 minor, 17 major], and 2 omphalocele minor patients were discharged against medical advice without intervention. The length of hospital stay ranged between 5 hours and 6 weeks (mean, 15.98±14.38; median, 10 days).

A total of 10 omphalocele major patients (29.4%) epithelialised and 7 others had healthy granulation tissue after 4-6 weeks and were discharged, but only 2 (11.7%) eventually returned for repair of ventral hernia. Fifteen patients (44.1%) with omphalocele major had primary closure (skin cover 4, fascial repair 11); however, 6 (40%) of these patients died from compromised respiration and progressive sepsis [Table 3]. One patient with enterocutaneous fistula complicating the omphalocele died of overwhelming sepsis after closure.

Postoperative complication rate was 32.1%, mainly due to sepsis 18 (51.4%), respiratory failure 18 (51.4%), wound infection 11 (31.4%), and diabetes insipidus 1 (2.9%) [Table 3]. Overall mortality was 30.4% from respiratory insufficiency post closure of ruptured omphalocele (5/11, 45.5%) and gastroschisis (3/7, 42.9%). The highest case fatality was among gastroschisis (4; 57.1%) and omphalocele major (11; 32.4%). Survivors were followed up for 6-14 months without events.

DISCUSSION

Increasing prevalence of omphalocele and gastroschisis has been reported from several regions of the world, and these have also necessitated a review of management approaches to reduce the morbidity and mortality.[1,4,8-10] True prevalence of the disorder is not known in Africa, especially in West African sub-region, possibly, because of non-availability of prenatal diagnosis and poor outcome of care post delivery.[2,7,11] Omphalocele is the most common anterior abdominal wall defect seen in our centre and other locations in Nigeria, though reports are scant.[2,3,11,12] There is a need for caution in the diagnosis of ruptured omphalocele (intrauterine or perinatal), which may be confused with gastroschisis that does not have a covering at all

Table 1: Age, gestational age and birth weight for patients with omphalocele and gastroschisis

Variables	Omphalocele (N=49)	Gastroschisis (N=7)
Age		
Range	5 -169 hours	12-74 hours
£±SD	27.5±11.3 hours	22.1±0.7 hours
Gestational age (weeks)		
Range	36-42	35-38
£±SD	39.2±0.7	36.6±0.5
Birth weight (kg)		
Range	2.4-4.2	2.3-3.3
£±SD	2.6±0.7	2.5±0.2
Maternal age		
Range	23-40	17-32
£±SD	28.5±5.9	25.3±6.8

Table 2: Places of delivery

Place of delivery	No. of patients	Percentage
Home	11	19.6
Private general practice clinic	23	41.1
Mission house	3	5.4
Teaching hospital	4	7.1
Primary health centre	15	26.8
Total	56	100



Figure 1: (a) Omphalocele major with intact sac showing liver and bowel. (b) Ruptured omphalocele with eviscerated stomach and intestine. (c) Intrauterine ruptured omphalocele with perforated foreshortened and oedematous bowel (arrow).

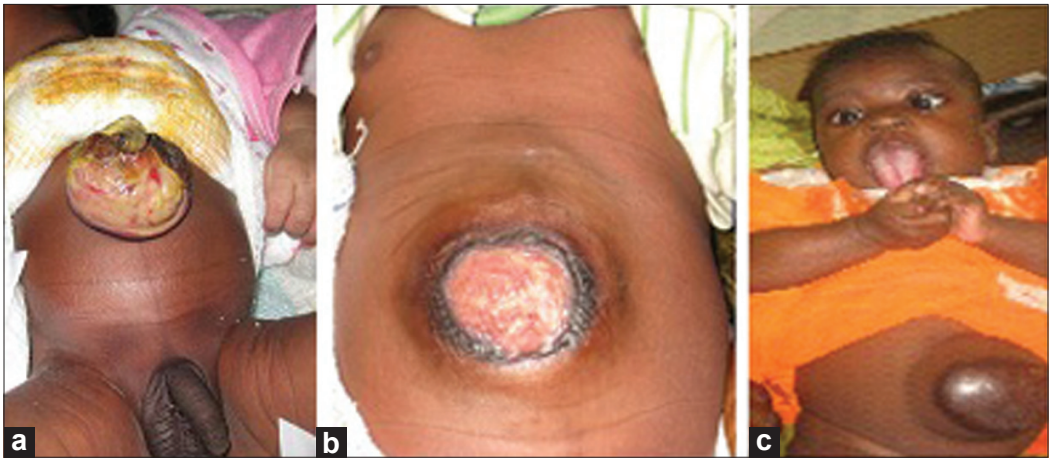


Figure 2: (a) Infected omphalocele, (b) good granulation tissue, (c) healed ventral hernia in a patient with Beckwith-Wiedemann syndrome.

because it is caused by intrauterine rupture of the right umbilical vein, leading to a defect in the abdominal wall adjacent to and not through the umbilical ring [Figure 1].[13] The irritation of the eviscerated bowel by the amniotic fluid makes the gut to be foreshorten, oedematous, and friable.

Table 3: Pre- and postoperative complications

Complications	*Pre-op (%)	Post-op (%)
Septicaemia	21 (37.5)	18 (54.1) [gastroschisis 3, omphalocele minor 5 (1 ruptured), omphalocele major 10 (4 ruptured)]
Ruptured Sac/exposed bowel	18 (32.1)	-
Hypothermia	39 (69.4)	-
Polycythaemia	3 (5.4)	-
Neonatal jaundice	7 (12.5)	-
Enterocutaneous fistula	3 (5.4)	-
Respiratory insufficiency	4	18 (54.1) [gastroschisis 1, omphalocele minor 6 (3 ruptured), omphalocele major 11 (6 ruptured)]
Wound infection	15	11 (19.6)
Polyuria ?diabetes Insipidus	-	1 (1.8)

*Multiple choice, Pre-op = pre-operative, Post-op= post operative

In spite of the obvious anomalies, there was delay in presentation (as shown by age at presentation) because many of these children were delivered at peripheral health facilities or at home in remote villages [Table 2]. This increased the morbidity and mortality, as factors like poor regulation of temperature and hydration status, improper care of the defect led to sepsis and vascular compromise of prolapsed gut during prolonged transportation [Figure 1], as observed in this and previous studies.[2,7,11,14] Many children may have died and were not presented at all because of inadequate measures taken to provide basic neonatal support before referral to our centre.[7,11,15] The final outcome of AAWD is significantly affected not only by postnatal conditions but also by the presence of additional structural and or chromosomal abnormalities. About 50-80% of

Table 4: Interventions and outcome in anterior abdominal wall defects

Outcome	Diagnosis	Treatment conservative	Operative	DAMA	Total (%)
Alive	Omphalocele minor	2	9	2	13 (23.2)
	Omphalocele major	11	9	-	20 (35.7)
	Gastroschisis	-	3	2	3 (5.4)
	Total	13	21		36 (64.3)
Dead	Omphalocele minor Omphalocele	-	2	-	2 (3.5)
	major	4	7	-	11 (19.6)
	Gastroschisis	1	3	0	4 (7.0)
	Total	5	12		17 (30.4)
DAMA	Omphalocele major	2	1	-	3 (5.4)
Total (%)		20 (35.7)	34 (60.7)	2 (3.5)	56(100)

DAMA = Discharge against medical advice

omphalocele may have other congenital anomalies, which may be life-threatening.[12,15-20] Detailed clinical, ultrasonographic scan, and radiological investigations should be done during postnatal resuscitation stage to detect these anomalies.[15] Investigations such as echocardiography and genetic screening were not done in this study due to lack of facilities and financial constraints. These investigations could guide the intervention and predict outcome. Currently, prenatal diagnosis of gastroschisis or omphalocele is recommended for complex examination and consultation at specialised centres so as to carry out accurate detection and appropriate classification of associated foetal anomaly for guiding the course of pregnancy.[19-22]

The high rate of antenatal ultrasonographic scan without diagnosing the AAWD in our study calls for a need to upgrade the routine antenatal ultrasound scan for prenatal diagnosis of this obvious defect so that in-utero transport to and possible intervention at specialist centres can help in reducing postnatal complications in the newborn.[19]

We are also challenged not only by the absence of facilities and personnel to make prenatal diagnosis but also by ethical issues that may arise from such practice. The single patient with gastroschisis detected by prenatal ultrasound at our centre had delivered in a private clinic and the infant was brought to our centre with gangrenous bowel and sepsis.

Primary repair of ruptured omphaloceles and gastroschisis without mesh, preformed silos, and mechanical ventilators as in this study should be avoided to limit poor outcome. We suspect that this closure caused an abdominal compartment syndrome, which resulted in compromised diaphragmatic excursion, decreased venous return via the inferior vena cava, and the consequent decrease in cardiac output as well as poor pulmonary, bowel, liver, and kidney perfusion, as reported in some series.[16,17,19]

Mabogunje and Mahour[23] observed that the patients' weight or size of defect and type of initial closure made no difference in the mortality of omphalocele. Perhaps, overwhelming sepsis could be a great detrimental factor in our study. Evidently, availability of preformed silastic silo, elective ventilation, and parenteral nutrition in developed countries has reduced the morbidity and mortality in many centres, and treatment is often completed in 2-3 weeks with good outcome.[18,24,25] In the absence of parenteral nutrition, we ensured regular haematocrit check, adequate blood and plasma transfusion, and early commencement of enteral feeding.

Non-operative management was adopted in many major omphaloceles because the peritoneal cavity was not adequate We used tulle-gras, silver sulphadiazine and/or honey dressing, and antibiotics with the intent of converting the defect to a ventral hernia [by formation of granulation tissue, which later epithelialised; [Figure 2] as done in other studies.[2,3,11,15,26,27] This conservative management takes about 1-2 months, with some done on ambulatory basis. However, in this study, many of our patients were admitted until full epithelialisation because the parents live in remote areas where the health facility often lack the provision of aseptic dressing materials and adequate care of the newborn. In our experience and other series, some patients were discharged, in frustration, by parents (against medical advice) who needed to look after the rest of the family and because of financial stress.[2,11] A study by Ameh *et al*,[7] revealed that improved outcome could be achieved in all neonates with omphalocele, irrespective of its size, by delayed closure after adequate stabilisation and treatment of infection. Some workers in developing countries have covered the hernias with skin (Gross method) to create a ventral hernia, which could be repaired later.[2,11,15] The success of this method has been limited at our unit. Two patients with impending rupture and the complete ruptured had skin

mobilisation to cover the omphalocele. They died a few hours after operation due to sepsis and respiratory failure, as elective ventilation was not available.

The overall survival in children with omphalocele and gastroschisis is now reduced to less than 10% in developed countries, especially in isolated cases without chromosomal anomalies or major organ malformation.[12,18,20,28,29] In this study, the mortality was 30.4%, with the highest case fatality among patients with gastroschisis (57.1%) and omphalocele major (32.4%). This is comparable with reports from the sub-region.[2,11] Many of these patients died due to infection and respiratory insufficiency. Only a few patients who had successful conservative treatment were brought back by the parents for secondary repair. This was also the experience at some other units in developing countries.[11,15]

There is still a big challenge in managing anterior abdominal wall defects in our sub-region. Basic care of the neonate and appropriate transport will reduce complications before presentation at the tertiary healthcare centres. Also, there is a need to improve on the neonatal intensive care facilities and personnels' skills, including aseptic nursing to provide succour to distressed parents presenting their children at our centre. Parents should be adequately counseled and provided enough information on the status of their infants to enable them to fully cooperate with the managing team and comply with management modalities. A study may be needed to confirm the microbiology sensitivity reports and potency of most generic antibiotics used in children with persistent overwhelming sepsis.

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