

REVIEW OF OMPHALOCELES (EXOMPHALOS)
IN
KENYATTA NATIONAL HOSPITAL
1976-1985

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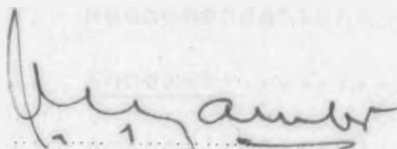
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PROF. J.M.KYAMBI

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SUMMARY:

A retrospective study of the omphlocele has been done. The study was carried out at Kenyatta National Hospital which is the referral hospital for the Republic of Kenya and the teaching hospital for the Faculty of Medicine of the University of Nairobi.

This study covers a ten year period from 1976 - 1984. A total of 34 cases have been studied. The yearly incidence has been static notwithstanding our high population growth.

Among the 34 cases there were 18 males and 16 females. Of the 34 cases there were 19 survivals and 15 deaths. 12 deaths (80%) among the 15 followed early surgery. Only 3 patients had surgery among 19 survivals. The major cause of death (6 out of 12) among the surgical cases was respiratory complications. Surgery had been performed within 1-3 days of birth.

Surgical management consisted of excision and repair of the omphalocele. Non-surgical regime entailed painting the omphalocele with mercurochrome (1 patient) or simply daily cleansing with antiseptics followed by sofratule dressing (18 patients).

Complication of the omphaloceles occurred in 4 cases only (Table 4). Malformations co-existing with omphalocele occurred in 12 cases of which 7 died. In our series non-surgical regime produced more survival (84.2%) 16 out of 19 cases.

REVIEW OF LITERATURE:

Literature on omphalocele has been reviewed with a view to bring out views as regards embryology, Pathogenesis incidence and various regimes of management. It is also the aim of the review to evaluate various Authors' results on various regimes of management and see how they compare with our results.

Earlier review of the literature was done by Jarcho (15) in 1937 which goes back to 1634. Prior to 1937, omphalocele as an entity has not appeared in literature. Jarcho (15) described omphalocele and discussed presentation, incidence, embryology, pathogenesis and modes of treatment both surgical and non surgical at the time.

Discussion of the embryology and pathogenesis has been done by de Vries (13) Ackerman (34), Williams (32), Soper (17), Holcomb (28) and Lamura (37). On treatment Grob (30) Soave (29) Ochola and Kyambi (22) describe non-surgical management while Gross (16) Eckstein (18), Moazam (23) and Schuster (24) discuss the various surgical techniques available for management of omphalocele. Othersen (27) discusses a new approach to surgical management where a pneumatic device is used to reduce the omphalocele followed by surgical repair.

Stringel (21) and Eckstein (18) discuss factors which influence the management of omphalocele both surgical and non surgical.

Welsebey (25) discusses a device for measuring the intra-abdominal pressure during the repair of the omphalocele.

King (19) and Touloukian (20) discuss the investigations which can be done to make intra-uterine diagnosis of omphalocele.

Total parental Nutrition in the omphalocele management, especially where gut function has been compromised by early rupture and subsequent infection is the subject of the paper by Filber (26).

Incidence of the omphalocele has been discussed by Jarcho (15), Beiley and Love (1), Soper (17) and Ackerman (34). The incidence varies from author to author ranging from 1:6,600 births in Jarcho (15) and 1: 1,860 births in Ackerman (34).

Most authors prefer early surgery. Early surgery in our series resulted in very high death rate. 12 deaths out of a total of 15 deaths had early surgery.

In contrast to world literature non-surgical management of omphalocele has been our regime of choice. Only those cases which must be subjected to surgery are managed surgically. This regime of non-surgical management has produced very encouraging results, there were only 3 deaths among the 19 patients on this regime.

AIM OF THE STUDY:

The aim of this study is to review the management of omphalocele in Kenyatta National Hospital during the period of 1976 - 1985. It is also the aim of the study to try and establish facts about:-

1. Presentation and incidence of the omphalocele in K.N.H.
2. The results of both surgical and non-surgical regimes of management.
3. Criterion for advocating one form of management in preference to the other.

When all these factors have been studied, a clear policy on management of the omphalocele should be made taking into account the following:-

- (i) Location of Paediatric Surgery Service in Kenyatta National Hospital.
- (ii) Transportation of patients from the periphery into K.N.H.
- (iii) The supportive care of the patients postoperatively with special reference to the intensive care unit.
- (iv) Materials available in this country for the managements of omphalocele.

It is felt that when all these aims are satisfied a recommendation should be able to be made to enhance survival of babies with imphaloceles in this country.

1.3 MATERIAL AND METHODS:

This is a retrospective study covering a 10 years period from 1976 to 1985.

MATERIALS:

Material for the study came from study of case records held at Kenyatta National Hospital records department. Retrieval of the case notes has not been as uneasy matter because before 1978 the paediatric surgical unit had not been established. All the omphaloceles were operated on by any surgeon on duty at the time of admission. The other problem was the classification of the omphalocele for records keeping purposes. Omphalocele has been moved from classification to classification even by the international classification index books held by our records department. It has been classified as umbilical hernia, as congenital umbilical hernia, gastroschisis, umbilical defect and as either omphalocele or exomphalos.

The most helpful retrieval of the case notes has been going through theatre records books, Theatre recovery room record notes, ward admission record books and by trial and error by various classification for notes retrieval held by the records department.

METHODS:

After the case notes were found, data was extracted as per data collecting form Annex A. Such data as name, race, location in Kenya (District) inpatient number, sex, birth weight, Birth defects both complications and malformations were extracted. Data pertaining to treatments given before surgery, age at surgery, surgical treatment done and the results were also extracted.

The data was then analysed and put into tables as laid out in the chapter on results. All the case notes were read thoroughly and all the factors contributing to death or the survival of the case recorded.

A total of 34 patients records were retrieved and analysed.

C H A P T E R 2

TABLE I:

TABLES OF RESULTS

YEARLY DISTRIBUTION OF OMPHALOCELES

YEARS	YEARLY INCIDENCE	ALIVE	DEAD	SURVIVAL RATE
1976 -	2	1	1	50%
1977 -	0	0	0	0%
1978 -	1	0	1	0%
1979 -	5	2	3	40%
1980 -	5	2	3	40%
1981 -	4	4	0	100%
1982 -	4	1	3	25%
1983 -	4	2	2	50%
1984 -	4	4	0	?100%
TOTALS	34	19	15	

TABLE 2:

SEX DISTRIBUTION

MALES	18
FEMALES	16
TOTAL	34

TABLE 3:

ETHNIC (TRIBAL) DISTRIBUTION

	NUMBER OF CASES	% OF TOTAL (347)	ADMISSIONS K.N.HOSPITAL
KIKUYU	18	52.94	33.9
KAMBA	6	17.65	19.8
LUHYA	5	14.71	9.2
LUO	1	2.94	17.2
EMBU	1	2.94	
MFRU	1	2.94	
SOMALI	1	2.94	19.8
KALENJIN	1	2.94	
TOTAL	34	100%	

TABLE 3:

ETHNIC (TRIBAL) DISTRIBUTION

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KAMBA	6	17.65	19.8
LUHYA	5	14.71	9.2
LUO	1	2.94	17.2
EMBU	1	2.94	19.9
MFRU	1	2.94	
SOMALI	1	2.94	
KALENJIN	1	2.94	
TOTAL	34	100%	100%

TABLE 4:

COMPLICATIONS

	No. Of Cases
Intestinal Obstruction	1
Gangrenous gut	1
Intussusception	1
Ruptured Sac	1
Total	4

TABLE 5:

CO-EXCISTENT MALFORMATIONS IN
INDIVIDUAL CASES WITH MALFORMATIONS

	No. Of Patients
Tetralogy of Fallot	1
Stenosis Ascending Colon	1
Beckwith Wideman Syndrome	1
Entero-Vitalline Fistula and Intussusception	1
Ano-Rectal Malformation and Epispadias	2
Ano-Rectal Malformation	1
Ectopia Vesicae and Gangrenous gut	1
Cleft Palate	1
Ano Rectal Malformation and Ectopia Vesicae	1
Down's Syndrome	1
TOTAL	11

TABLE 5:

CO-EXISTENT MALFORMATIONS IN
INDIVIDUAL CASES WITH MALFORMATIONS

	No. Of Patients
Tetralogy of Fallot	1
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Ano-Rectal Malformation and Epispadias	2
Ano-Rectal Malformation	1
Ectopia Vesicae and Gangrenous gut	1
Cleft Palate	1
Ano Rectal Malformation and Ectopia Vesicae	1
Down's Syndrome	1
TOTAL	11

TABLE 6:

INDIVIDUAL MALFORMATIONS

	NUMBER OF CASES	% OF TOTAL CASES (34)
Ano-Rectal Malformations	3	8.8%
Ectopia Vesicae	2	5.9%
Down's Syndrome	1	2.94%
Entero Vitelline Fistula	1	2.94%
Beckwith Wideman syndrome	1	2.94%
Cleft Palate	1	2.94%
Fallot's Tetralogy	1	2.94%
Stenosis of Ascending colon	1	2.94%
TOTAL	11	

TABLE 7:

DEATHS

(A) MANAGEMENT REGIMES:

	NUMBER OF CASES
Surgical	12
Non Surgical	3
Total	15

(B) ASSOCIATED MALFORMATIONS:

	NUMBER OF CASES
With Malformation	8
Without Malformation	7
Total	15

TABLE 8:

CAUSES OF DEATH

(A) SURGICAL CASES

	NUMBER OF CASES
RESPIRATORY	6
CARDIAC	4
BURST ABDOMEN	1
FAILURE TO THRIVE	1
TOTAL	12

(B) NON SURGICAL CASES

	NUMBER OF CASES
CARDIAC FAILURE	3
TOTAL	3

TABLE 9:

(A) LIVE CASES

	NUMBER OF CASES
SURGICAL	3
NON SURGICAL	16
TOTAL	19

(B) LIVE CASES WITH ASSOCIATED MALFORMATIONS

	NUMBER OF CASES
WITH MALFORMATIONS	5
WITHOUT MALFORMATIONS	14
TOTAL	19

TABLE 10

METHODS OF TREATMENT

SURGERY	15
NON SURGICAL	19
TOTAL	34

RESULTS:

The results of the 34 cases studied are analysed graphically in the tables 1 - 10. The following can be inferred from these results.

1. Presenting Complaints:

The presenting complaints are from the parents. The infant is normally very comfortable. The parents complain about the hernia at the umbilical region at birth. The neonate may also have other complaints. With improved diagnostic methods (19) and (20), the diagnosis should be made before birth.

2. Family History:

History of omphalocele appearing as a familial disease has not been elicited. This is in keeping with world literature.

3. Past Medical History:

Except in the four cases seen in 1985 no medical history pertaining to omphalocele had been recorded. In the 4 cases no history of previous malformations in the family was recorded. No history of hydramnios was recorded. History of drugs, radiation and food eaten would be interesting but was not recorded either.

4. Incidence: (Table I)

In spite of fast population growth in Kenya the yearly incidence of omphalocele appears to be constant. 1985 shows an early start with 4 cases in the 1st quarter.

5. Ethnic distribution - (Table 3)

The results are a reflection of the normal hospital admissions except that Luos though being one of the major population groups in the hospital (17.2%) have very few case, 1 in those 10 years. A better method of displaying this data would be by locality of origin. This would be important when considering teratogens both industrial and resident to a place.

6. Sex - (Table 2)

Both sexes are represented equally
male:female, 18:16.

7. Complications encountered (Table 4)

Only 4 cases had complications of their omphalocele. Complication is that medical problem superimposed on the omphalocele.

The complications were :-

1. Intussusception -1
2. Gangrenous gut -1
3. Intestinal obstruction-1
4. Ruptured Sac -1

There was only 1 death (33%). The patient is one with the gangrenous gut discovered at operation of the omphalocele. The neonate had to have excision of the sac, resection and anastomosis of the gut and primary repair of the abdomen. This patient also had ectopia vesicae. He died 3 days

post operatively. The other three survived. The second patient developed obstruction in the ward on the 3rd day of non surgical management. At operation viable gut was found and reduced. The omphalocele was excised and repaired primarily. The patient was discharged on the 15th postoperative day.

The third patient with intussusception developed it on the 18th day while on conservative treatment. This was reduced and the omphalocele was excised and repaired in one stage. A concomitant enterovitelline fistula, discovered at operation, was also excised. The neonate was discharged on the 24th post operative day.

8. Congenital malformation encountered Tables 6,7,9

The commonest malformations were found in the intestines. Out of the 11 individual malformation only 3 were not from the gastrointestinal tract. These were the Tetralogy of Fallot one case, Cleft palate one case and one case of Down's Syndrome.

Table 5 shows that there were 11 patients with malformation and in 4 cases there were more than one malformation at the same time in the same patient.

9. Effect of Congenital Malformation on the outcome of treatment:

Table 7, 8, 9

Out of the 34 cases there were 15 deaths and 19 survivals. 7 of the patients who died and 5 of the living ones had other congenital malformations. It is observed that among those who died, of the 12 who died after surgery, 5 had malformations. Apparently malformations increased mortality among the surgical patients.

10. Treatment (Table 7, 8, 9, 10).

Out of the 34 patients 19 had non-surgical regime of treatment producing 16 survivals. The three deaths died suddenly of cardiac arrest. Of the 15 who had surgery, 12 died, there were only 3 survivals. It appears the early surgery is associated with higher mortality. Among the 12 dead, 4 had intact omphalocele membranes with no complications. These sacs were excised and the defect covered with dura graft. These grafts got infected causing severe peritonitis. One of the patients had 3 burst abdomen, in weekly intervals. This frustrated the mother so much that she tore the patients notes before deserting the patient who eventually died.

The major causes of death was cardio pulmonary in 10 patients. Comparing the two regimes of management non-surgical regime produced higher rate of survivals.

11. Type of Surgery:

This was normally excision of the omphalocele sac and closure. In 4 cases this was difficult and dura graft had to be used to cover the defect. The four died postoperatively.

12. Type of Non-surgical Treatment:

The neonate was put in an incubator for warmth and the sac painted with mercurochrome in one case while the other 18 had simple dressing with sofratulle which is a light weight dressing containing Framycetin sulphate 1%. This is changed daily and the sac cleaned with antiseptic solution like Eusol. Only 3 patients died under this regime. 1st case was under 2500 gms and had failure to thrive. This would have benefitted from Total parenteral Nutrition if it was available here. The 2nd patient died suddenly of cardiac causes. In addition the patient had Ectopia vesicae and epispadias. The 3rd patient had Down's Syndrome and also died suddenly. The inference we can draw from the various regimes of management is that non-surgical treatment in our series yields better results.

CHAPTER 3

DISCUSSION:

3.1 DEFINATION: The name omphalocele according to "Dorland's illustrated medical dictionary" (6) is a synthesis from two Greek words "omphalus" Greek for umbilicus and "Kele" = Greek for 'hernia'. Hence omphalocele means "Umbilical hernia".

A true umbilical hernia contains abdominal visceral herniating through a large Umbilical ring. This hernia is covered by skin and peritoneum.

Omphalocele on the other hand is still herniation of abdominal viscera through a large umbilical ring. The herniating viscera is covered by peritoneum, part of the original intra embryonic coelomic Epithelium (de Vries (13)) and a membrane derived from the distended cord. This membrane consists of Amniotic membrane externally (13) and Wharton's Jelly in between. The whole sac is a clear membrane which allows visibility of the viscera enclosed within. Cruising on it are found umbilical vein cranially and two umbilical arteries caudally.

According to Fasana (5), Gross (16), William (32), Boyd (33) and de Vries (13) the membrane contains in the Wharton Jelly, lots of fibroblasts and a mesh of collagen fibres. The interstasis are filled with mucopolysaccharides ground substance.

This composition is very important when considering non-surgical regime of management of the omphalocele as when denatured by drying, the collagen fibres Shrink (Gross 16) reducing the content of the omphalocele into the adominal cavity at the same time giving a gentle traction to the abdominal wall to encourage growth. Grob (30) Joy (31).

So omphalocele is essentially a herniation of abdominal viscera into the umbilical cord. de Vries (13), Fasana (5) Jarcho (15), Sober (17) and Gross (16) do not agree this is a hernia. They describe it as an arrest of embryonic stage of development when the gut developed outside the abdomen - an eventration - as this gut has never been inside the abdominal cavity and then herniated subsequently nor does it reduce and then herniate like most hernias do before they become irreducible due to obstruction. This is a more acceptable explanation considering the embryology of the omphalocele which is the subject of the next few pages.

3.2 EMBRYOLOGY OF OMPHALOCELE:

Most of the embryologists (Fasana (5) de Vries (13), Jarcho (15), Gross (16) and Soper (17) agreed that omphalocele (exomphalos) is an arrested embryonic developmental stage. Though resembling a hernia omphalocele content has never been inside the abdominal cavity and then later herniated out. They further agreed that between 4 - 12 weeks of intrauterine life mid gut of the embryo grows very fast. The growth is faster than the elongation of the embryo (Fasana (5) Soper (17)). This is also the stage of fast growth of the liver which pushes the gut out of the abdomen. This is made worse by the curvature of the embryo ventrally. The mid and the hind gut then herniate into the umbilical cord base forming an umbilical cord sac where the guts continues to grow. At about 12th week the embryo when about 44 mm, the gut returns into the abdominal cavity. This is encouraged by the rapid growth of the caudal part of the embryo creating a large abdominal cavity in the pelvis and lower abdomen. The embryo also tends to straighten at this stage. The return of the gut is associated with 270° rotation of the small gut anticlockwise. Failure of the gut to return is normally associated with malformation of the gut and persistence of the mesocolon due to failure of fusion of the dorsal mesocolon to the posterior abdominal wall & peritoneum producing attachment of the colon to the posterior abdominal wall.

When this process of reduction of the gut from the umbilical cord sac to the abdominal cavity fails then an omphalocele is formed from about 12 weeks of intrauterine life.

It is this finding of gut in a sac outside the abdomen through a large umbilical ring which made some authors call this an umbilical hernia.

When the membrane gets ruptured and lost in the intra uterine life the condition is mistaken with another condition called Gastroschisis. In this congenital condition the gut herniates out of the abdominal cavity through a hole, not covered by a membrane, mostly at the left side of the umbilical ring leaving the umbilical cord attached at its normal position. When the omphalocele membrane is ruptured during the intrauterine life, umbilical vessels must be preserved or else the foetus will die.

These vessels will be attached to the umbilical ring which is a distinct entity. It consists of a fibrous ring covered internally by peritoneum and externally by embryonic ectoderm, towards the abdominal wall, and Amniotic ectoderm towards the umbilical cord (Boyd J. 33). There is an area of transitional epithelium between the two.

3.3 PATHOGENESIS OF OMPHALOCELE

There is a general concensus that omphalocele is due to failure of the gut to return to the abdominal cavity after 12 weeks of intrauterine life or when the embryo is about 44 mm.

The area of controversy is how this happens. Some authors (Fasana (5) and Gross (16) Soper (17) agree that it is not known how this happens. However they agree it could be associated with:-

- Failure of abdominal cavity to enlarge or
- May be due to failure of the abdominal wall to grow.

There are authors who offer other explanations. As quoted by Gross (16) Alfield (1899) suggested that it is the persistence of the omphalomesenteric duct 'tethered' to the umbilical cord sac which prevents the gut from reduction. This exclusion of the gut from the abdominal cavity may cause failure of the abdominal wall to grow due to lack of stimulus as the gut stays out of it, a form of disuse atrophy. This reasoning was based on the fact that Meckel's diverticulum in its various forms, fibrous band, Fistula, Sinus or a Cyst commonly associated with omphalocele 9% in Herbert T (18), and 4.5% Gross R. (16), Enbom (35) who studied human and opossum embryos suggest that contraction of the longitudinal muscles in the omphalomesenteric artery

may help to withdraw the loops of gut into the abdominal cavity, failure of which could result in an omphalocele, de Vries (13) implies that omphalocele is caused by failure of the abdominal wall somites to grow and then meet at the midline. Another explanation is persistence of a large embryonic body stalk which keeps the rectus abdominis muscle from fusing at the midline.

The arrest of the abdominal wall growth and failure to come together at about 44 mm size embryo can be explained as a generalised failure of fusion of the ventral side of the embryo as it folds ventrally. This could explain the associated anomalies of midline deficiency like in (i) ectopia cordis due to abnormality of sternum, sternal edges of ribs and pericardium. (ii) Ectopia vesicae due to failure of anterior or ventral wall of the urinary bladder. This ventral midline failure with the associated anomalies in thorax and caudal part of the abdomen is called by Cautrel (36) and Towne (14) midline failure syndrome. In the midline failure syndrome the omphalocele may contain herniating organs like heart, liver, stomach, spleen, pancreas and at times gall-bladder. These organs are usually normal as they were never involved in the failure affecting the abdominal wall.

Towne (14) has observed that in very large omphaloceles the recti muscles are inserted more laterally at the costal margin. This is in keeping with large embryonic connecting stalk explanation where the stalk is supposed to keep the rectus abdominis muscles from uniting at the midline

3.4 INCIDENCE:

Incidence of omphalocele has been reported
by Jarcho (15) 1937 as 1: 6,600 deliveries,
Beiley and Love (1) as 1: 6,000 deliveries
Soper (17) as 1: 4,000 births and
Ackerman P (34) as 1: 3,200 in England
1: 1,860 in U.S.A.

It has not been possible to work out incidence in
Kenyatta National Hospital. The neonates who were treated
in Kenyatta National Hospital were referrals from other
hospitals. Small omphaloceles from these centres are treated in these
centres conservatively. The deliveries in Kenyatta National
Hospital are normally of ladies with complications referred
from the other hospitals so they do not form a good
sample of deliveries from where incidence can be calculated.
Notwithstanding it is important that incidence be worked
out in forward planning especially where government has
put a lot of effort in having accurate census and recording of
deaths and births.

3.5 CLASSIFICATION OF OMPHALOCELE:

Various methods have been used to classify omphalocele. Some authors Bailey and Love (1) classify them into major and minor. This is in relation to insertion of the cord and size. Minor have cord at the apex of the sac and are small. They contain intestines and at times Meckel's diverticulum. Major are large mostly contain liver and the cord inserted caudally. Other authors classify them according to size. These fall into two classes large - when more than 5 cm in diameter and small - when less than this as sighted by Towne (14), Stringel (21), Ochola and Kyambi (22) and Schuster (29). Others classify them according to the viscera present Liver having a bad prognosis. Towne (14) calls them "Hepatomphalocele" when they contain the liver. In our set up all these have been tried initially depending on surgeon who saw the patient. In the last three years our imphalocelles were classified only as large or small; 5 cm diameter still being the cut off. The largest omphalocelles are about 15 cm. This is practically about the anatomically possible diameter or else beyond this, it will cease to be imphaloccele because it will take up all the abdominal walls and include bladder and part of the chest in it.

Classification of the omphalocele has not been found to influence prognosis. Any classification should take into account that a very large omphalocele may have very small neck. In these cases the viscera which has come out through the small umbilical ring will still have problem being returned into abdominal cavity. If the neonate has been fed, the gaseous distension of the intestines may predispose it to intestinal obstruction, as in one case in our series. Eckstein (18) sites cases of empty omphalocele sac. This should be a joy to the surgeon there being no gut to reduce. Eckstein (18) noted that when umbilical cord has cranial insertion to the sac (10%) omphalocele was usually associated with agenesis of the hindgut, ectopia vesicae, and in 2% had abnormal lower limbs.

Most authors agree, Eckstein (18), Stringel (21), Towne (14) and Ochola and Kyambi (22) that the size of the omphalocele sac does not influence mortality significantly. The factors which influence mortality and should be included in classification are birth weight, associated congenital defects and rupture of the omphalocele sac before surgery. It is a comfort for those bent on non-surgical management of omphalocele that repair of the omphalocele sac which is torn is done and Stringel (21) reports survival after the repair and subsequent non-surgical management.

3.6 NATURAL HISTORY OF OMPHALOCELE AND NON-SURGICAL
TREATMENT:

Omphalocele starts its life between 6 and 12th week of intrauterine life. At birth it is found as a transparent avascular sac through which the enclosed viscera can be seen. Cruising on it are one umbilical vein cranially and two umbilical arteries caudally. Gross (16), Soper (17), Halocomb (28) and Soave (29).

Only rarely does omphalocele rupture before birth 2.94% in our series. If rupture has not occurred at birth it could then happen after birth due to rough handling of the omphalocele. A few days after birth (3 - 4 days) the omphalocele membrane starts to change and becomes opaque. Later on dries and cracks as it shrivels up. At times especially when wet it becomes infected, necrotic, smelly and may rupture causing eventration of the gut and severe peritonitis with cellulitis of the abdominal wall. When this process is going on at the surface of the omphalocele at the peritoneal side the membrane is invaded by granulation tissue starting from the edge of the sac. In small sacs this would take a few days and even if the original membrane crusts off the granulation tissue which has now turned into epithelial tissue holds on (see photos).

In our set up a 15 days old granulation tissue has been found strong enough to contain the gut even when the neonates cries. (see pictures).

Later on the granulation tissue is invaded externally by skin epithelial cells from the edge of the omphalocele and the whole sac is covered by skin converting the original omphalocele to a ventral hernia.

Understanding of this natural history is important when designing a model for non-surgical treatment of the unruptured omphalocele.

It is imperative that when non-surgical omphalocele is undertaken one has to find a way of:-

- (i) Preventing rupture of the omphalocele
- (ii) Prevent infection of the omphalocele sac.
- (iii) Keep the membrane long enough for epithelialization to take place.
- (iv) Plan to repair the subsequent Ventral hernia
- (v) A criteria for selection of patients to be included in the non-surgical management is also essential.

Before 1940 when Penicillin came into general use infection was very important and conservative management of the omphalocele was disastrous. The best treatment was surgery and the earlier it was the better. Soper (17) reports that

mortality rate was 12% when operation was within 12 hours and 31% when within 24 hours and 62% when operated within 48 hours. After this it was even higher.

At this age of good antibiotics, infection can be controlled. To prevent rupture of the membrane omphalocele should be anticipated even before birth. Robert J. (20) had found maternal ultrasonography accurate in diagnosing congenital malformations especially when combined with good history and measurement of Alfa-Foetal Proteins in the amniotic fluid Charles (19). The best time is at 33 weeks gestation. With ultrasonography Omphalocele was one of the malformations easily diagnosed. The decision should be made this early on how to protect the sac from rupture. Most centres have had only one or two rupture before birth. In our series only one had ruptured in 34 cases 2.94%. After the child is born this exposed sac should be dressed with wet saline gauzes to keep it wet and to prevent rupture. It should be emphasized that this thin membrane especially in large omphalocele where loops of gut liver and even stomach may be included, forms a very poor heat insulation for the body core. The infant as a rule suffers from hypothermia very fast. To prevent this most authors suggest covering the imphalocele sac with warm gauze soaked in saline and covering the rest of the body especially lower part in a plastic bag. Akcerman (34), Towne (14) and Ochola and Kyambi (22).

In early literature where conservative management was advocated Jaricho (15) Holcomb (28), Soave (29) Grob (30), the omphalocele sac was cleaned with spirit then painted with 2% aqueous mercurochrome. The mercurochrome denatures the membrane and hardened it. It also made it difficult to be infected by bacteria. This unfortunately dried up and craked exposing the granulation tissue below it. With luck by the time it dropped off granulation tissue and strong epithelia had formed and then covered with skin taking one to three months Soave (29). The only contraindication to mercurochrome method is the risk of mercury toxicity as reported by Fagan et al(39)Stringel (21).

In our set up only one case had mercurochrome painting afterwards a very simple dressing of sofratulle has been very successful. This has the advantage of keeping the sac sterile and also moist. When it is moist it stays longer, over four weeks and by the time it sloughs off the underlying granulation tissue and epithelialization is strong. As for the repair of the ventral hernia, this poses very little problem. In the authors series six patients have had their ventral hernias repaired. They range in age between 1 month and 2 years. All had well formed abdominal wall and the hernia was easy to repair in one stage.

Their hospital stay during the repair ranged between 5 days and 15 days. As regards criteria most authors Gross (16), Holcomb (28), Soave (29) and Grob (30), advocate that patients for conservative management should be those with complications and other congenital malformations which make surgery risky. Patients with very large omphaloceles even if they have no complications are also recommended for conservative treatment.

This was also the principle, "womb to theatre" practiced before 1978 in this hospital. Most of the operated patient died miserably following the surgery. One had multiple burst abdomen. The other three had intact omphalocele membrane which was excised and then the abdomen repaired with dura graft. All these patients had infection of the dura graft and all died with severe peritonitis.

As from 1979 only those patients who needed very urgent surgery were operated. Those were the cases which needed reduction of intussusception one case, colostomy for ano rectal malformation two cases, release of abstraction - one case colostomy for stenosis of ascending colon - one case. All others who did not need urgent surgery were treated conservatively with sofratulle dressing. Most of these patients healed well to had their central hernia repaired later.

3.7 SURGICAL MANAGEMENT OF OMPHALOCELE:

Surgical repair of the omphalocele is as old as the surgery. Surgeons of all the ages have been plagued by this congenital condition whose mortality to date has stayed high. What has changed over the ages has been the ability to diagnose omphalocele before birth. Robert (20) and Charles (19).

Another field which has changed is in improvement of management of sepsis especially the introduction of penicillin in 1940 and availability of prosthetic surgical materials for covering of the omphalocele. With all these improvements omphalocele still has high mortality in most centres.

Jarcho (15) and Schuster (24) have reviewed history of omphalocele surgery over the ages. Celsus and his follower Paulus all recognised umbilical hernia and suggested reduction of the content and ligation of the sac in older children. The same treatment was not advocated for infants with the same condition. Francos' treatise on hernia in 19th century does not deal with umbilical type.

Ambroise Pare described congenital umbilical hernia as early as 1634, but inspite of all these literature very little was written on omphaloceles before 19th century.

Aribat reviewed older literature in 1901 and found only 9 cases reported before 1800. The earliest mention is by Scultetus in 1634 though no surgical treatment was attempted.

It is William Hey who in 1803 described 3 cases of "hernia congenita umbilicalis". Before that, records of umbilical hernia never mentioned it as congenital. Pott Richer and Mauriceau in 17th century described umbilical hernia and treatment was advised as bandaging only though no success was guaranteed as "the success of it is very rare".

In 1807 Sir Asthey Cooper in his treatise on umbilical hernia mentions that some infants are born with umbilical hernia and described the first recorded surgical management on congenital umbilical hernia. He reports one case where James Hamilton reduced the hernia tied the sac at the base and then "cautiously opened it". The edges of the abdominal wall was then brought together with two silver pins and some adhesive straps".

The sac was allowed to drop off and the cure was said to have been complete in a few days. The child was still well 8 months later.

Before this operation Hamiltons had several other neonates with congenital umbilical hernia but had not attempted operation as he considered them as "strictly desparate".

Samuel Bard in America in 1819 mentions children born with congenital umbilical hernia. He recommends treatment by "reduction, straping with adhesive plaster and moderate

pressure by a proper bandage". Some treatment was advised by Scanzoni in 1955. It appears then that early treatment of the omphalocele (congenital umbilical hernia) was non-surgical and reduction and compression was the method of choice. It is only Hamilton who attempted surgery of the omphalocele. This apathy on treatment was mainly due to the poor record of survival of these infants.

Pybus 1922, Jarcho 1937, Fresher, 1926 and Cullen 1916 all advocate early surgery in omphaloceles.

They advocate excision of the sac and closure of the abdominal wall. The operation should be undertaken as early as possible after birth. When the omphalocele was simple containing ^{gut} only radical operation gave very good results. Aribat in 1901 operated 68 cases with 47 (69%) recoveries. Altpeter in 1900 operated 91 cases with 69 (75.8%) recoveries.

When the sac contained liver or other viscera results were not as good. Out of 45 patients operated only 11(24.5%) recovered. N.M. Dott in Edinburgh Medical Journal in 1932 put the thinking of the time very well "The child should pass straight from the womb onto the operating table."

This one stage repair of omphalocele posses its own problems. It is no wonder that those neonates with compound omphaloceles fared so badly with only 24.5% survival rate. Most of the omphalocele surgery has as it evolved from those early days of 19th century, had to find solutions to improvement on survival on the compound omphaloceles.

One stage repair technique solves only one problem the closure of the abdominal wall defect when possible. The closure brings in new problems which were not there before surgery. All the problems stem from the effort to place the herniated viscera into the abdominal cavity, a place it has never ever been and which does not have the space to receive them as it has never developed it. In one stage repair except in few cases this entails forcing the viscera into an underdeveloped abdominal cavity. A series of problems then emanates from this increased abdominal pressure.

Gross (16), Soper (17), Towne (14), and Stringel (21) list complications emanating from increased abdominal pressure as:-

- (1) Displacing the diaphragm into the chest causing respiratory distress.
- (2) Pressure on the inferior vena cava causing reduction of cardiac return, circulatory collapse, multiple haemorrhages in the gut and oedema of the legs.
- (3) Intestinal Kinking causing obstruction and paralytic ileus.
- (4) Herniation of the stomach into the thorax causing respiratory distress, hiatus hernia and gastroesophageal reflux.
- (5) Budd-Chiari phenomenon with ascites due to Kinking of the inferior venacava at the diaphragm level as the liver is manipulated back into the crowded abdominal cavity.

Any surgeon should try and overcome these problem as death from respiratory and cardiac collapse is the major cause of mortality within the first 6-48 hours after omphalocele surgery. This was 29.7% in Eckstein's (18) series. In our series of the 15 patients who died, 6 (40%) patients had respiratory associated deaths, and 4 (26.6%) had sudden death which could be circulatory collapse. Combined respiratory and cardiac related deaths accounted for 86.6% of the deaths in the surgical cases.

Respiratory embarrassments seem to be a very common cause of death. This is not strange as a neonate up to 10 days old breaths with the diaphragm according to Baur (7), Gross (3), Swenson (4), Bren TCK (9), Richardson (10), Respiration vol. I (11), Respiration vol. II (12). Any surgery which interferes with the diaphragmatic respiration makes it very difficult for the neonate to breath. It has been found hence very necessary to have a very good set up for respiratory support where omphaloceles are operated. Eckstein (18). Towne (14) suggest that the respiratory support can be a lengthy problem lasting for months at times. There were 12 respiratory problems out of 53 cases (22.64%) Towne (14). One of the 12 (8.3%) needed a tracheostomy. 4 cases out of 9 (44.4%) treated for congestive cardiac failure had pulmonary hypertension as the primary cause.

Towne (14) also mentions a condition when liver has to be pushed back into the abdominal cavity. The manipulation of the liver causes angulation of the inferior vena cava at the diaphragmatic level causing demonstrable Kinking when cardiac catheterization is done. This causes ascites secondary to Budd-Chiari phenomenon. This has been supported by Waldinar (1977) and Carlton as quoted by Towne (14).

To solve these problems surgeons have developed various methods of surgery to make omphalocele surgery safe. The sole aim has been to devise a method which does not raise the intra abdominal pressure after the abdomen is closed. Other lines have been to find an equipment which can be used firstly for research purposes to define what pressure is acceptable without causing the complications mentioned earlier.

Welsley et al (25) came out with a safe pressure of 20 cms of water. Othersen et al (27) in their pneumatic device for gradual reduction of the omphalocele using air pressure found that 50 cms of water pressure was safe.

One none prosthetic solution to the increased abdominal pressure after closure was offered by Buchaman (36) who advocated splenectomy and partial hepatectomy up to 20 gms to allow closure. This method should be condemned as splenectomy before 2 years of life has been associated with severe infections later in life. La Mura (37) and Morgensten (38).

As regards surgical technique staged closure has been found to be the safest. The staged closure refers to the abdominal wall. To achieve this the earliest method perfected by Gross (16) in 1940 involved excision of the omphalocele, dissection of the skin from the abdominal wall as far posteriorly as possible, reduction of the omphalocele content and then closure of the skin. Subcutaneous fat may be closed also if intrabdominal pressure allows. Abdominal muscles were not approximated.

The infant would then grow up with a ventral hernia which could be repaired later in life. There are basically two problems with this method of surgery. Firstly since the abdominal wall is left free, one is essentially substituting omphalocele sac with skin and so the gut continues to grow under the skin cover while the abdominal wall receives no stimulus to grow. When one goes to repair the ventral hernia problem of closure would still surface again. Release incisions at the flanks were used to try and achieve closure of the ventral hernia Denison (2). Second problem was adhesion of the intestines to the raw skin surface needing sometimes excision of the gut to release the adhesions' Towne (14).

Gross (16) solved his problem of adhesions by leaving the omphalocele sac intact cutting into the skin just near its margin then dissecting the skin as before and then closing it over the sac thus leaving a peritoneum covered membrane still covering the intestines. Since the gut was not handled, incidences of ileus were rare. Hilcomb (28) 1961 still practise Gross's technique. Burying of infected membrane was a source of problems with infection at times.

More current surgical techniques make use of artificial membranes to cover the omphalocele contents after the sac has been excised. The membranes are sutured to the abdominal wall and the growth of the abdominal viscera produces enough gentle tension to stimulate growth of the abdominal wall making later closure of the ventral hernia possible and easy.

The common artificial membranes used are Teflon Mesh Moazom (23) Schuster (24) and Filter (26), and Silastic Sheet Othersen (17).

The original use of the prosthesis by Moazam (23) was to bridge the abdominal muscles with the Teflon (Polytetrafluoethylene) Mesh and then with the prosthesis in place to approximate the mobilised skin over it. This was sort of improving of the quality of the ventral hernia over Gross (16) whose skin cover only did not stimulate the growth of the abdominal muscles for easy closure of the ventral hernia later on. The prosthesis could be removed later when the abdominal wall has grown and repair of the ventral hernia done or the prosthesis could be left in place for a long time.

The Mesh is then invaded by fibroblasts and then incorporated into a pseudomembrane. Some of the meshes have been left indefinitely. Towne (14) reviewed this method of treatment and found that infection, fistula formation and severe adhesion of the gut requiring gut resection complicated the surgery. In one patient 8 surgical procedures were required with a 274 days stay in hospital and \$122,000 bill.

Schuster (24) improved on this technique. The improvement was on prevention of the adhesions and shortening of the hospital stay. The Teflon Mesh was covered by silastic to make it less liable to produce adhesions. To reduce the chances of adhesions formation even further the Teflon Mesh was then lined by a non reactive and impermeable sheet of polyethylene or silicone rubber. This kept the abdominal viscera away from the Teflon Mesh. Like the Teflon this was in two pieces each for each side overlapping at the midline. The Teflon without tension was then sutured at the midline and then where possible skin was sutured over it. When this was not possible an Island of Mesh without skin was left to be dressed with sterile dressing. On the 4 - 6th day the patient was taken back to theatre and the Mesh which was now lax, due to the stretching of the abdominal wall and growth of the muscles, trimmed and again without tension the procedure was repeated. This was repeated again and again till the abdominal wall could be approximated at the midline without undue tension.

The average stay in the hospital was 24-63 days. Mortality was reduced to 27.2% and a remarkable survival of 5 patients with antenatal ruptured omphalocele membrane compared with only 1 case before.

Othersen (27) has come up with a device like a belljar which is made of Dacron and lined with silastic sheet. When it is sutured to the omphalocele edge the area between the bell and the silastic sheet is filled with air at 50 mm water which acts as a pneumatic reduction device of the omphalocele facilitating closure of the hernia later. The procedure can be done under local anaesthetic and the reduction procedure takes about 7 - 10 days after which the repair of the omphalocele can be undertaken.

Most of the surgeons agree that supportive procedures are very important for success of good omphalocele surgery. The prenatal diagnosis (19) and (20) is important if preparations are to be made to receive the neonate born with omphalocele. After birth presence or absence of the omphalocele sac is important. When absent the quality of the gut is compromised as they are meconium stained, oedematous and matted together. Soak in 50,000 units of fibrinolysin in 50 mls of water helps to lyse the fibrin exudate, Othersen (27). Intact membrane should be covered with wet soaks and neonate protected from the inevitable hypothermia.

Total parental nutrition is important especially where gut is compromised usually in ruptured omphaloceles, Filber (26). This is important since the neonate has to be starved for surgery. For easy surgery the gut needs to be decompressed by gastrostomy or Nasal gastric suction. In our series Nasal gastric suction has been sufficient.

Most surgeons prefer immediate surgery, some in consideration of cost Towne (14). The preference is for primary closure and where this is difficult staged repair. This could become obsolete if devices like the pneumatic reduction are perfected since the gentle traction causes the abdominal wall to grow as the omphalocele is reduced for easy one stage repair.

Advocates of early surgery argue that when the abdomen is opened one is able to inspect the whole gut and ascertain whether other operable abnormalities do exist. The commonest abnormalities arising from the gut are atresia, obstruction or agenesis. If there is a method which can lower the mortality rate without necessarily having to open the abdomen like the pneumatic reduction device it ^{should} be encouraged as, the anomalies can be investigated by non-invasive methods as in other neonates with no omphalocele.

Anaesthesia is important in neonate surgery. The best anaesthesia has been found to be non paralysing anaesthesia as toneless abdominal muscle can not be used to judge the intra-abdominal pressure reached at closure of the abdomen.

CHAPTER 4

4.1. CONCLUSION:

There is still controversy on pathogenesis of omphalocele. Though review of the literature suggests that most surgeons prefer surgical management of the omphalocele and the same excuted as early as possible our results seem to suggest that our patients survive better under non-surgical treatment.

Neonatal surgery has its own problems and supportive facilities like early diagnosis, transport, theatre and intensive care unit with good respiratory support are important. We have encountered problems in postoperative period because supportive equipments such as respirators and incubators may not be operative and Parental nutrition is not always available. Since respiratory support is vital availability of functioning ventilators for long periods is mandatory, Temperature control is also important and working incubators^{are} essential.

Knowing our limitations non-surgical treatment where practicable should be the treatment of choice. Infact all the omphalocele patients in our set up should be programed for conservative management until other conditions dictate, early surgery.

This entails a good incubator, and simple moist dressing like sofratulle. Early surgery where essential may be performed but simpler methods like pneumatic reduction before surgery could be advocated since they do away with the need for prosthetic sheets and multiple operations.

In surgical management primary repair should be the aim. If this is not possible then Gross's method of mobilising skin cover over intact sac would be a simple straight forward procedure leaving the ventral hernia for later repair.

4.2. RECOMMENDATIONS

This study has been very stimulating in that it has unearthed lots of controversy on management of omphalocele. The controversy ranges from pathogenesis, classification, nomenclature and even management. Without getting involved in the controversy over Embryology and pathogenesis one could concentrate on management which I think is beneficial to the patient.

Suffice it to say that in present state of our medical services non-surgical management appears to yield the best results. Surgery should be restricted to those cases which must be operated and simple technique is what we need. To achieve this end I would recommend that:-

1. It is important to have good obstetric service where good history and management of pregnancy can be done so that those cases who by behaviour of the pregnancy, like hydramnios are likely to have congenital malformations, should be delivered in a hospital where the malformations can be adequately treated.

2. As regards omphalocele when discovered at birth the sac or the exposed intestines, when sac is ruptured, should be covered by wet sterile swabs and a firm bandage put on.
3. The neonate should then be kept warm possibly in an incubator. If no incubator is available cover the baby with warm cotton wool and a blanket.
4. A nasal gastric tube should be passed to decompress the abdomen and when necessary I-V fluids should be started and administered at the appropriate rate.
5. Thorough medical examination of all the systems should be done to rule out any complication or associated anomaly.
6. The omphalocele should be classified and then a decision made whether surgery is needed or non-surgical management is the treatment of choice. All ruptured omphaloceles would benefit from immediate surgery.
7. The neonates on conservative management should receive clear fluid for a few days to keep the gut undistended then later start on² dilute milk feed.

8. The neonate should be warmed in an incubator or wrapped in a warm blanket.
9. The neonate should be observed very closely for infection of the omphalocele membrane and respiratory system as any cough may contribute to the rupture of the sac.
10. Plan for repair of the ventral hernia can be done when the child is big enough but preferably before school age.
11. As regards surgery the simplest procedure should be done. Gross procedure of covering the omphalocele with skin should be tried as a method of choice.

5.1 ANNEXA

FORM USED FOR COLLECTING DATA
FROM CASE NOTES.

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PHOTOGRAPHS:



15 days
old



45 days
old

REFERENCE

1. Bailey and Love's
Short Practice of Surgery
18th Edition 1981. E.S. Livingstone Ltd.
2. W.M. Dennison
Surgery in infancy and childhood
Seven Edition 1967.
3. Gross
The Surgery of Infancy and Child Hood- Saunders
June 1955.
4. Oruar Swenson 1962.
Paediatric Surgery (second Edition)
Appleton Century - Crofts MC.
- 5: F. Fasana
Text book of Medical Embryology. Normal and
Abnormal Development.
Kenya Literature Bureau 1980.
6. Dovland's. Illustrated Medical Dictionary
24th Edition 1965.
7. A. Robert Bauer M.D.
American Journal of Diseases in Children 1940
vol. 60: 1342 - 1350
Respiration in Newborn Infants.
8. James Mason Brown 1962 802 - 803
"Surgery of Childhood"

9. Brown T.C.K. 1979
"Anaesthesia for Children" pg. 6.
10. Richardson P.P. and John J.H.
"Neonatal Surgery"
1970 pg. 35 - 36. Butterwoths London.
11. "Respiration" vol I 1965
American Physiological Society pg. 213.
12. "Respiration" vol. II 1965
American Physiological Society Pg. 1334.
13. Pieter A de Vries
"The Pathogenesis of Gastroschisis and
Omphalocele".
Journal of Paediatric Surgery vol. 15 No. 3:
245 - June 1980.
14. Barbara H. Towne and George Peters
"The Problem of the Giant Omphalocele".
Journal of Paediatric Surgery 1980 vol. 15:
543 - 548.
15. Julius Jarcho, M.D. F.R.C.S.
Surgery Gynaecology and Obstetrics vol. 65
Nov. 1939 No. 5: 593 - 600.
"Congenital Umbilical Hernia."

16. Robert Gross M.D. and James B. Blodget M.D.
Omphalocele (Umbilical Hernia) in the
Newly Born.
Surgery, Gynaecology and Obstetrics 1940
vol. 71: 520 - 527.

17. Robert T. Soper M.D. and Edarward W. Green.
"Omphalocle".
Journal of Surgery, Gynaecology and Obstetrics
Oct. 1961. 501 - 508.

18. Herbert and Echstein
"Exophalos". A review of 100 cases.
British Journal of Surgery 1963 vol. 50:
405 - 410.

19. Chairles R. King M.D. and Gerald H. Prescott D.M.O.
"Amniotic Fluid Alpha-Foetoprotein Elevation
with Fetal Omphalocele and a Possible mechanism
for its occurence."
American Journal of Obstetrics and Gynaecology
vol. 130 (3) Feb. 1978.

20. Robert J. Touloukian and John C.
"Maternal Ultrasonography in the Antenatal
Diagnosis of Surgically Correctable Fetal
Abnormalities".
Paediatric Surgery vol. 15: No. 4 August
1980 373 - 377.

21. G. Stringel and R.M. Miller
"Prognostic Factors in Omphalocele and
Gastroschisis".
Journal of Paediatric Surgery vol. 14 No. 5:
Octo. 1979.
22. Ochola-Abila P.M. Med. J.M. Kyambi M.D.,
F.A. and S.M. Barrack M.D.
"The management of Giant Omphaloceles"
The Proceedings of the Association of
Surgeons of E.A. 82.
23. Farhat Moazam, Bradley M., Rodgers and J.N. Tabert
"Use of Teflon Mesh for Repair of Abdominal
wall Defects in Neonates".
Journal of Paediatric Surgery vol. 14 No. 3
(June) 1977.
24. Samuel R. Schuster M.D. F.R.C.S.
" Anew Method for the Staged Repair of Large
Omphaloceles".
Surgery, Gynaecology and Obstetrics Oct. 1967
vol. 125: 837 - 850.
25. John R. Welsley, Robert Drougowski and Annold G.
C. Coran.
"Intragastric Pressure Measurements:
A guide for Reduction and Closure of the
Silastic Chimney in Imphaloce and Gastroschisis".
Journal of Paediatric Surgery 1981 vol. 16 No.3
(June). 264 - 270.

31. Jay L. Grosfield, M.D. Lilian Dawes and Thomas R. Weber.
"Congenital Abdominal Wall Defects: Current Management and Survival."
Surgical Clinical of North America (Oct.) 1981
vol. 61 No. 5: 1037 - 1049.

32. P.L. William C.P. Wnedell-Smith
Basic Human Embroyology
Second Edition
Pitman Medical and Scientific Publication
1969 Pg. 66 - 67.

33. Boyd J.D.
Human Placenta
Cambridge W. Heffer and Sons Ltd. 1970.

34. Ackerman P.
Congenital Malformation
Chapter XV (215 - 231) Excerpta Medica.

35. Ebbon G.
Der Repositionsmechanismus des Physiologischen Nabelbruches bei Saugetieren und beim Menschen.
"Morphol." Jahn 1938, 82:271.

36. Buchanan R.W. and Cain W.L. 1956
"A case of a complete Omphalocele".

37. Joseph La Mura et al.
"Splenorrhaphy for the Treatment of Splenic Rupture in Infant and Children".
Surgery (May) 1977 vol. 8 No. 5: 497 - 501.
38. Loen Morgensten
"Techniques of Splenic Conservation".
Arch. Surg. 1979 vol. 114 449 - 453.
39. Fagan D.G. Pritchard J.S. Clarkson T.W.
"Organ Mercury Levels in Infants with Omphalocele Treated with Organic Mercurial antiseptics."
1977 Arch. Dis. Child 52: 962 - 964.