



Fetal ascites in cloacal malformations—a red flag

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Abstract

Introduction Cloacal malformation is a rare anomaly that remains a diagnostic challenge prenatally, despite the current advances in ultrasonography and MRI. This condition can in some, present with isolated ascites or with other findings, such as a pelvic cyst or upper urinary tract dilatation. In a minority, the ascites may be progressive, questioning the role of antenatal intervention.

Methods We report on ten patients that have been identified from our Cloaca database between 2010 and 2022.

Results The presence of ascites was associated with extensive bowel adhesions and matting, leading to a challenging initial laparotomy and peri-operative course.

Conclusions Antenatal finding of ascites in newborns with cloacal malformations should raise a red flag. The surgeon and anaesthetist should be prepared for the operative difficulties secondary to bowel adhesions and the higher risk of haemodynamic instability at the initial surgery. An experienced team at initial laparotomy in such patients is vital.

Level of evidence: II.

Keywords Antenatal · Ascites · Hydrocolpos · Cloaca · Adhesions · Stoma

Introduction

Ascites is a rare finding denoting an underlying pathology in more than 90% of patients. Causes include chromosomal anomaly, intrauterine infection and congenital malformations affecting the gastrointestinal, genitourinary, or cardiovascular systems [1]. Cloacal anomaly occurs in 1:50,000 live births and involves the drainage of the bladder, vagina, and rectum into a common channel to the exterior through a single perineal orifice. This is caused by a failed separation of the cloaca at 4–6 weeks of gestation into a urogenital sinus anteriorly and a hindgut posteriorly [2]. The previously

known concepts of embryogenesis have more recently been refuted and lean towards the principle of subdivision of cloaca being a passive process, namely, differential growth, and spatial realignment of tissues [3, 4]. Ascites in cloacal malformations has been noted in more than 20% of cases (Fig. 1) [5].

Cloacal anomalies are frequently associated with other manifestations of obstructive uropathy which includes the presence of hydrocolpos (Fig. 2A), unilateral or bilateral hydronephrosis (Fig. 2B), oligohydramnios and calcified meconium in the peritoneum. Ascites in a fetus with a cloaca is caused by the flow of urine from the bladder into the common channel, then back up through the vagina, uterus and fallopian tubes into the abdominal cavity. Such directional flow has been demonstrated in patients with both a cloacal anomaly and isolated urogenital sinus during postnatal genitograms [6]. Chronic irritation of the tubal mucosa by the outflow of urine and meconium may ultimately lead to tubal blockage and regression of the ascites for the majority, but in a small subset, the ascites is progressive, leading to chronic irritation of the abdominal cavity [7, 8].

We report ten patients in whom ascites caused extensive intra-abdominal adhesions from chronic peritoneal irritation

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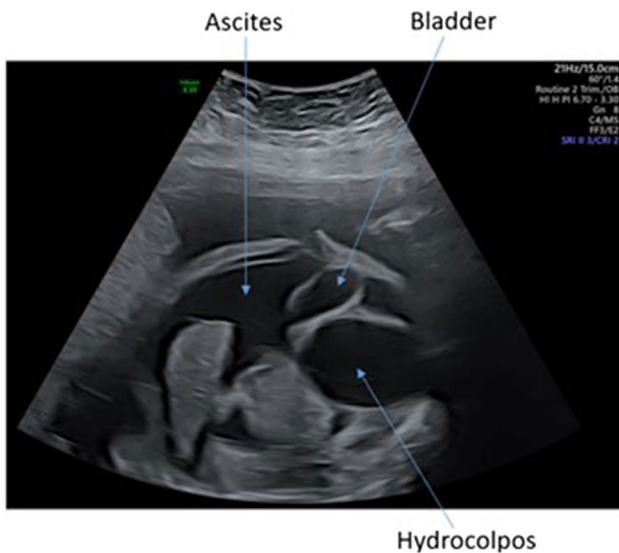
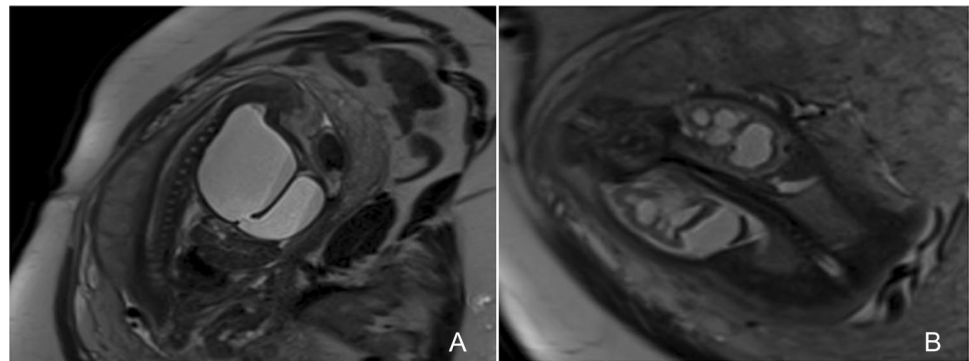


Fig. 1 Ultrasound 28-week gestation

leading to a challenging neonatal procedure with haemodynamic instability.

Fig. 2 Fetal MRI. (A) Hydrometrocolpos. (B) Bilateral hydronephrosis



Methods

Patients with prenatal ascites were identified from our Cloaca database (2010–2022). Data were collected regarding antenatal history—presentation, amniotic fluid index (AFI), upper urinary tract dilatation (UTD), hydrocolpos, and interventions. Post-natal course included gestation and condition at birth, mode of delivery, immediate postnatal interventions, findings at initial laparotomy, bowel and urinary diversions, cardiorespiratory support and renal outcomes were all noted.

Results

During the study period we had 69 new patients with the diagnosis of a cloacal malformation. Ten were identified with prenatal ascites. Only one reached term and the others were prematurely delivered. All ten patients presented in the third trimester with a pelvic cystic mass, and severe fetal ascites. Four had prenatal interventions of whom two had paracentesis and the other two abdomino-amniotic

Table 1 Summary of prenatal findings and interventions

	Presentation (weeks)	AFI	Upper urinary tract	Fetal intervention
Patient 1	28/40	Normal	B/L UTD	AAS Shunt (28/40)
Patient 2	31/40	Normal	Solitary kidney + UTD	–
Patient 3	28/40	Oligohydramnios	B/L UTD	–
Patient 4	26/40	Oligohydramnios	B/L UTD	Paracentesis only
Patient 5	26/40	Normal	B/L UTD	–
Patient 6	28/40	Normal	Solitary kidney	–
Patient 7	26/40	Polyhydramnios	Solitary kidney	Paracentesis only
Patient 8	31/40	Normal	Solitary kidney	AAS Shunt (31/40)
Twin 1				
Patient 9	25/40	Normal	B/L UTD	–
Twin 1				
Patient 10	30/40	Polyhydramnios	B/L UTD	–

Table 2 Summary of postnatal findings and outcomes

	Gestation at birth (weeks)	APGAR	Intervention immediate post-natal	Common channel length (cm)	Intra-operative findings	Diversions	Need for resuscitation	Current eGFR	Follow-up/current age
Patient 1	34 NVD	Good	–	4	Widespread dense adhesions, inflammatory exudate, and pseudo-cyst	Right transverse colostomy	Haemorrhagic shock, Blood transfusion, On-table cardiac arrest	Normal	12 years
Patient 2	35+2 Em-LSCS	Poor	–	3	Extensive adhesions with matted bowel loops	Sigmoid loop colostomy	No	Normal	6 years
Patient 3	35+1 Em-LSCS	Poor	2×Abdominal drains (160 ml aspirated)	3.5	Dense bowel adhesions, Significant blood loss during adhesiolysis	Loop ileostomy (Exact site uncertain)	On-table cardiac arrest, Blood transfusion	Normal	4 years
Patient 4	31+1 Em-LSCS	Poor	–	3 Posterior Cloaca	Fixed bowel loops, several adhesions, significant blood loss	Transverse colostomy	Blood transfusion	Normal	2 years
Patient 5	32+6 Em-LSCS	Poor	Abdominal drain	5	Thickened peritoneum, matted bowel loops	Transverse colostomy	No	Normal	5 years
Patient 6	39+2 Em-LSCS	Poor	–	2.5	Matted bowel loops Re-laparotomy due to bowel obstruction with adhesional volvulus	Ileostomy	No	Normal	20 months
Patient 7	31+2 Em-LSCS	Poor	–	Posterior Cloaca 3	Extensive adhesions	Descending colostomy, Vesicostomy	No	Normal	1 year
Patient 8	32 NVD	Poor	–	3	Extensive adhesions with matted bowel loops	Ileostomy	No	Normal	7 years
Patient 9	33 Em-LSCS	Good	–	Posterior Cloaca 2.5	Dilated colon, large ascites	Sigmoid loop colostomy	No	Normal	5 years
Patient 10	34 Em-LSCS	Poor	–	3	Colon difficult to mobilise, widespread adhesions	Transverse colostomy	No	Normal	9 years

shunt (AAS). All except one had UTD and were bilateral in those that had both kidneys. The findings are summarised in Tables 1 and 2.

Of the 69 patients, 25 had hydrocolpos (36.2%). All 10 patients with fetal ascites had a pelvic cyst in the third trimester consistent with hydrocolpos. Three of 10 had posterior cloaca. In the remaining 15 with hydrocolpos but without

ascites the course was uneventful in comparison with the former group. None required adhesiolysis or had a stormy perinatal period. Our study cohort had common channels greater than 2.5 cm and 80% of them were more than 3 cm.

Case 1

Fetal intervention in the form of AAS was performed due to the severity of ascites, with some reduction in the volume towards the end of pregnancy. Labour was induced and the baby was born by vaginal delivery with no requirement for respiratory or cardiovascular support prior to transfer to our institution. Laparotomy was technically difficult and long due to several bowel adhesions and eventually a right transverse colostomy was performed. Haemorrhagic shock led to an on-table arrest requiring 6 min of resuscitation. Blood, platelets and FFP were transfused. On transfer to the neonatal intensive care, she suffered another cardiac arrest requiring 30 min of resuscitation and high frequency oscillatory ventilation for the next 5 days before being extubated successfully. There was transient acute kidney injury but currently, her eGFR is 128 ml/min/1.73 m². Abdominoperineal PSARP and TUM was carried out months later, where widespread adhesions were encountered but this was more easily addressed without any hemodynamic compromise.

Case 2

Increasing ascites in utero led to preterm delivery. VACTERL association diagnosed with oesophageal atresia with trachea–oesophageal fistula, duodenal atresia, solitary left kidney, and ventricular septal defect. Gastrostomy formation and duodenal atresia repair were done, along with a sigmoid loop colostomy in the first 24 h of life. Extensive adhesions with matted bowel loops were noted in the initial procedure. No active resuscitation measures were required. Perineal approach TUM and PSARP was carried out later. No particular difficulty noted during the procedure, but colostomy reversal was challenging due to limited bowel mobility needing adhesiolysis. In a few days, a colcutaneous fistula was apparent which on further assessment revealed the colon anastomosis had strictured. She then had a defunctioning ileostomy, TPN and redo surgery later.

Case 3

The baby was born in poor condition necessitating intubation and resuscitation at birth. Two abdominal drains were placed due to ongoing poor saturation despite FiO₂ of 100%.

Laparotomy on day 1 revealed massive hydrometrocolpos and dense bowel adhesions. Attempted adhesiolysis resulted in significant blood loss leading to a cardiac arrest and cardio-pulmonary resuscitation for 2 min. There was a good response to packed red blood cells and FFP transfusion. A loop ileostomy was fashioned; however, due to patient instability, the exact site or segment was uncertain. Abdominoperineal approach to PSARP and bladder neck closure with mitrofanoff formation was carried out soon after. A very long common channel and absent urethra prompted the above decision. Minimal adhesiolysis was required on this occasion and during ileostomy closure.

Case 4

Antenatal aspiration of ascites was performed but no shunt was left in situ. The baby was delivered by emergency caesarean section due to distress, in poor condition, and needed immediate intubation. Pulmonary hypoplasia with pulmonary hypertension required Nitric Oxide and Sildenafil. Intraoperatively, on day 2, matted, fixed bowel loops and several adhesions were found. Further dissection to identify structures was not possible; however, a transverse colostomy was finally isolated and formed. Packed red cells and platelets were administered during surgery due to significant blood loss. Later a perineal approach PSARP and TUM was carried out with no difficulty.

Case 5

Immediate postnatal intubation with ventilation and an abdominal drain was inserted. Along with a single perineal opening, multi-organ malformations were identified consistent with a VACTERL association. Oesophageal atresia repair, tube vaginostomy and diversion stoma were all carried out in the first week of life. At laparotomy, thickened peritoneum and extremely matted bowel loops made it quite challenging to identify the appropriate loop for diversion but eventually a transverse colostomy was achieved. A common channel of 5 cm and hardly any measurable urethra prompted a bladder neck closure and a mitrofanoff a few months later. Some minor adhesiolysis was required during the above reconstruction and at stoma closure.

Case 6

Baby was born in poor condition with severe abdominal distension and difficult ventilation, which lead to emergent laparotomy. A Meckel's diverticulum was excised but due to

technical difficulties with matted bowel loops and adhesions, an ileostomy was fashioned. Unfortunately a few months later she developed intestinal obstruction and underwent a laparotomy confirming adhesive volvulus requiring 10 cm of small bowel resection. She went on to have a perineal approach PSARP and TUM at around 2 years of age. Very minimal adhesiolysis was required at stoma closure.

Case 7

Rapidly filling fetal ascites noted and paracentesis provided a transient response. Baby was born by LSCS at 31 weeks as the ascites was progressively worsening leading to hydrops. At birth severe abdominal distension was noted. Intraoperatively there was matted bowel and widespread adhesions with a large amount of free fluid. Prolonged surgery for adhesiolysis, and a descending colostomy was fashioned. Later a perineal approach PSARP and TUM was performed with no adhesiolysis required at stoma closure.

Case 8

Severe ascites at 31-week gestation, in association with bilateral hydrothorax and skin oedema in a dichorionic diamniotic twin 1 led to an AAS placement to decompress the abdomen. Babies were delivered by emergency caesarean section due to distress of twin 1, born in poor condition and required immediate intubation for respiratory distress secondary to severe abdominal distension. The single functioning right kidney was hydronephrotic, associated with hydrocolpos, and a catheter was passed into the common channel. Left kidney was a MCDK. Laparotomy on day 2 of life revealed extensive adhesions with matted bowel loops. Adhesiolysis was not attempted, and an ileostomy fashioned, 15 cm from the ileo-caecal junction. A long common channel with absent urethra, small bladder and spinal dysraphism prompted a bladder neck closure, augmentation cystoplasty, mitrofanoff and PSARP. Minor adhesiolysis was required at the above reconstruction but stoma closure was uneventful.

Case 9

Twin gestation with hydrocolpos and fetal ascites were noted at 25 weeks of gestation. Pre-term delivery at 32 weeks occurred, by emergency C-section due to early signs of twin-to-twin transfusion. Twin 1 was born in good condition but developed severe abdominal distension and bilious vomiting

in the first 18 h after birth. At laparotomy, severe ascites was noted with 250 ml aspirated, dilated left colon with some bowel adhesions culminated in a loop sigmoid colostomy. She later underwent a perineal approach PSARP and TUM. Stoma closure required minor adhesiolysis.

Case 10

Hydrocolpos and ascites were detected at 30 weeks of gestation, with bilateral hydronephrosis. Baby was born in poor condition leading to intubation and ventilation. Oesophageal atresia and TOF, were repaired soon after birth along with a transverse colostomy at the same time. During the laparotomy, she remained stable but the abdominal intervention was technically challenging due to dilated loops of bowel with several widespread adhesions, making it extremely difficult to identify the appropriate loop of large bowel for diversion. Transverse loop colostomy was then fashioned followed by an abdominoperineal approach employing PSARP and TUM. Minor adhesiolysis was required both at reconstruction and stoma closure.

Discussion

Cloacal anomaly is a rare malformation affecting 1:50,000 births. It is confirmed post-natally with the finding of a single perineal orifice, but antenatal diagnosis is frequently made facilitated by advanced ultrasonography and MRI imaging [2]. Despite these advances antenatal diagnosis can still be challenging.

Livingston et al. reported on 50 female patients born with a cloacal anomaly over an 11-year period. In their study only 6% were accurately diagnosed antenatally, although 54% had an antenatally detected abnormality on ultrasound [9]. They reported three signs that can raise the suspicion of a cloacal anomaly. In their series 52% had an intra-abdominal pelvic cystic mass representing a hydrocolpos, 49% had urinary tract abnormalities such as hydronephrosis and 18% had dilated bowel loops. Of these 26% had oligohydramnios which can occur if there is a bladder outflow obstruction [5]. They theorised that the presence of oligohydramnios might contribute to the difficulty in diagnosing the pelvic anomalies of the fetus. In the above series, 7 (14%) had associated ascites of which 6 spontaneously resolved [9].

Ascites is generally a sign of underlying pathology, and it is essential that a diagnostic work-up is undertaken to investigate for other causes, including cardiac, chromosomal, haematological anomalies, infections, and structural anomalies [1]. In cloacal anomalies urinary ascites

is hypothesised to be caused by urine draining into the peritoneal cavity through the fallopian tubes [7].

Another possible cause could be the intermittent rupture and resealing of the urinary bladder [9]. This may be similar to ascites resulting from in-utero bladder perforation secondary to PUV [10, 11]. It has been hypothesized that urinary extravasation may have a protective effect on renal function due to a “pop-off” phenomenon. A literature review of cases found 75% preservation of renal function with pre-natally detected urinoma caused by a lower urinary tract obstruction [12–14], though there are some papers questioning this effect [15]. Urinary ascites can also be caused by urine extravasation from the upper urinary tract rupture but is rare. The extra-peritoneal collection can lead to peritoneal fluid via transudation [16]. It is interesting to observe that in all our patients with immediate postnatal drainage in some form or the other has preserved renal function eventually.

Adams et al. proposed that the presence of significant outflow resistance to urine due to presence of a hydrometrocolpos and a long common channel may lead to aberrant drainage of urine and meconium [6]. In our series eight patients had a long common channel of ≥ 3 cm in addition to the presence of hydrometrocolpos which is consistent with this hypothesis. The ascites is usually transient as the urine or meconium causing chemical irritation eventually leads to obstruction of drainage via the fallopian tubes [7]. Livingston et al. reported on the transient nature of ascites in their series in 6 of 7 patients with cloacal anomalies [9]. This was not found in our series, as all our patients had progressive non-resolving ascites and only one happened to have some reduction of the ascites volume towards the end of pregnancy. The persisting leak of urine and meconium causes extensive irritation of the peritoneal cavity consequently leading to inflammation of the bowel wall, adhesions, and matting. Meconium has been visualised extruding through the fallopian tubes into the peritoneal cavity with no evidence of intestinal perforation in a term female neonate with cloaca [8]. Meconium peritonitis was suspected with plaque-like calcification in the flanks. In our series, none of our patients had frank meconium peritonitis. However, Motoi et al. reported on one neonate with a cloacal anomaly and progressive urinary ascites with a hostile peritoneum precluding formation of a diverting colostomy for up to 14 days postnatally. Pathology of a specimen taken from the wall of an intra-abdominal cavity revealed a foreign body reaction to meconium. The child was alive at 6 months of age but there was no comment about renal function in their report [17].

Urinary ascites can also occur in other genito-urinary anomalies, including persistent urogenital sinus. Although this is extremely rare it has been reported in several publications [18, 19]. In their report Loganathan et al. confirmed back flow of urine through the hydrometrocolpos and

fallopian tubes into the peritoneum on post-natal genitography. There were no associated renal or urinary anomalies in their patient [18].

The association of oligohydramnios and pulmonary hypoplasia is well-known. Several authors have documented the relationship between increasing ascites and worsening oligohydramnios. They considered them as poorer prognostic factors with reports of neonatal deaths in these cases [2, 7, 19, 20]. Petrikovsky et al. reported on 3 babies with foetal ascites due to cloacal anomalies. A favourable outcome was only noticed in 2 of these in whom the ascites resolved in utero [7]. This is also true in our series as two of our patients that had reduced amniotic fluid volume were born in poor condition requiring respiratory support at birth.

Despite the poor prognosis noted with increasing ascites, intervention is not without hazards. Morikawa et al. [18], reported intra-uterine demise in their patient following 4 episodes of paracentesis followed by an insertion of an AAS. Despite resolution of the ascites, the baby was still-born 12 h after the shunt insertion at 30-week gestation. They hypothesized that the baby succumbed due to an acute circulatory collapse [20]. Two of the patients in our series had a placement of AAS due to extensive ascites. Case 1 had successful resolution of the ascites for 5 weeks prior to the shunt becoming blocked causing distress requiring emergency delivery. We assume that the shunt may have become blocked causing re-accumulation of the ascites. Cases 3 and 5 did not undergo an intervention for ascites; however, immediately after birth, it was decided to insert an abdominal drain as a life-saving procedure. Cases 4 and 7 had paracentesis in-utero but no shunt was inserted, with progressive clinical deterioration of the fetus resulting in emergency caesarean section. Finally, Case 2, 6, 9 and 10 did not undergo a fetal shunt placement or abdominal drain post-natally as an urgent laparotomy soon after birth was done instead. Prenatal intervention to facilitate drainage and reduce the compartment syndrome is controversial. Given that the development of the renal metanephros and the lung canalicular phase are both complete in the second trimester any procedure with this intent would have very little benefit for organ development and confer increased risks of prematurity. However, the degree of initial renal and respiratory compromise emphasises the importance of having a plan for perinatal or early postnatal decompression in these circumstances. Figure 3 shows a newborn with severe abdominal distension on respiratory support.

All our patients had a hostile peritoneum during their initial laparotomy. Three of the patients suffered significant blood loss due to the requirement for extensive adhesiolysis to identify the anatomy. Two of those patients required massive blood transfusion and had a very turbulent intraoperative and postoperative course with on table



Fig. 3 Newborn with severe abdominal distension due to ascites, on respiratory support

or post-operative arrest requiring resuscitation. Three of the ten children had extremely difficult dissection and adhesiolysis and the surgeon opted for a diverting ileostomy. This is similar to what has been reported by Motoi and colleagues [15]. Interestingly, in our cohort of 59 cloaca babies without fetal ascites, the first laparotomy has generally been straight forward with no evidence of peritoneal irritation, difficult dissection, or any blood loss.

Bischoff and colleagues proposed a guide to prenatal counselling for cloaca and cloacal exstrophy. They recommended that all cases with antenatally diagnosed hydrocolpos on imaging should be delivered at a tertiary centre. Parents were made aware of the need for early decompression of the vagina and the need for a diverting stoma [20]. Our group is the first to report on the challenging management of babies born with progressive ascites due to cloacal anomalies. For this reason, we propose that during the prenatal counselling parents should be made aware of the inherent risk of a volatile post-natal course in the presence of progressive ascites as shown in our case series. It is crucial they understand the initial laparotomy might be very challenging due to the potential bowel matting and adhesions brought on by the peritoneal reaction to both urine and meconium. This creates an increased risk of blood loss and hemodynamic instability. Patients falling into this group should be managed by an experienced team in anticipation rather than the most junior person on call.

Another point would be the greater potential for this patient group being the long common channel variety. Although this observation still warrants further studies, we believe that it may be alluded to during the counselling

sessions. This obviously would require further confirmation postnatally but may be suggested by the progressive nature of the ascites. The combination of the anticipated intra-abdominal adhesions and the nature of a long common channel anomaly would, therefore, make future reconstruction more challenging.

The antenatal history of severe progressive ascites in a newborn with a cloacal malformation is a red flag. Surgeons and anaesthetists should be prepared for the operative difficulties secondary to bowel adhesions and the higher risk of hemodynamic instability at the initial surgery. The association between progressive ascites and the possible presence of a long common channel should be suspected. These would be best served with antenatal counselling and planning the perinatal period. The authors also believe the ascites may potentially provide a reno-protective effect due to the pop-off mechanism.

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Data availability Data available on request from the authors.

Declarations

Competing interests The authors declare no competing interests.

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