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ORIGINAL ARTICLE

Persistent cloaca with fetal ascites: clinical features and perinatal management

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Abstract

Purpose Fetuses with persistent cloaca are known to develop urine or meconium backflow into the abdominal cavity caused by obstruction of the common channel, thus leading to fetal peritonitis with fetal ascites. We analyzed the impact of prenatal fetal ascites on postnatal clinical features and management.

Methods This retrospective single-center cohort study was conducted to compare the perinatal parameters of patients with isolated persistent cloaca who were born and treated at our hospital between 1991 and 2021. The clinical features and management of those with and without fetal ascites were compared.

Results Among the 17 eligible patients, fetal ascites were recognized in seven. The occurrence of fetal ascites was significantly related to preterm birth, higher birth weight z-score, birth via emergency cesarean delivery, low Apgar scores at 1 min and 5 min, higher C-reactive protein levels at birth, longer duration of oxygen administration, the need for a urinary drainage catheter at initial discharge, and shorter neonatal hospital stays.

Conclusions The postnatal management of patients with persistent cloaca with fetal ascites differed significantly from that of patients without fetal ascites. For patients with unexplained fetal ascites, magnetic resonance imaging may be helpful for determining the definite diagnosis of persistent cloaca.

Keywords Persistent cloaca · Fetal ascites · Prenatal diagnosis · Urine drainage · Magnetic resonance imaging

Introduction

Persistent cloaca is a rare disease that occurs in approximately 2 out of every 100,000 individuals. This condition comprises the convergence of the rectum, vagina, and urinary tract into a single common channel that opens at the urethral meatus, thus representing the most severe type of anorectal and urogenital malformation [1, 2]

Partitioning of the cloaca into a urogenital sinus anteriorly and a separate hindgut posteriorly begins at 4 weeks of gestation and is complete by 6 weeks of gestation [3, 4]. Although there are great internal and external anatomical variations in patients with persistent cloaca, aberrant urine drainage usually occurs because of the abnormal confluence of the three systems. Obstruction of the outlet causes not only ureterohydronephrosis, which may impair renal dysfunction, but also persistent cloaca, which causes the urine and meconium to flow into the vagina, resulting in hydrometrocolpos or backflow into the abdominal cavity through the fallopian tubes [5]. This backflow of urine and meconium may cause the development of chemical peritonitis in the fetus [6].

Although several case reports have described the relationship between the existence of fetal ascites and outcomes of patients with persistent cloaca [3, 5–8], no comprehensive case–control studies have been conducted. Furthermore, there have been several case reports of persistent cloaca with fetal ascites. Warne et al. reported one case involving neonatal death and another case involving chronic renal failure [4]. Petrikovsky et al. reported a case involving full-term delivery, which is rare in other reports, and intrauterine growth restriction [7]. Nigam et al. stated that large fetal ascites can also lead to diaphragmatic compression, pulmonary hypoplasia, and fetal death [5]. Morikawa et al. argued that fetal

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ascites and oligohydramnios indicate a poor prognosis and discussed the effectiveness of fetal therapy [3]. However, no comprehensive case–control studies have been published.

Therefore, we compared prenatal and postnatal parameters of patients with persistent cloaca with and without fetal ascites to evaluate the impact of prenatal fetal ascites on perinatal clinical features and management.

Methods

Medical records of patients with persistent cloaca born at our hospital between April 1991 and March 2020, were retrospectively reviewed. This included all patients with a postnatal diagnosis of persistent cloaca and with or without a prenatal diagnosis. The prenatal diagnosis was determined by an obstetrician using fetal ultrasonography and a radiologist using fetal magnetic resonance imaging (MRI). These imaging modalities described all the findings of persistent cloaca, especially hydrocolpos, hydrometros, hydronephrosis, ureterohydronephrosis, double vagina, and double uterus. The fetal MRI strongly indicated persistent cloaca based on the findings of reflux of urine into the rectum and the rectum running through the vaginal septum of the double vagina. Patients with severe airway or cardiac anomalies were excluded from the study.

The patients were divided into one group with ascites and one group without ascites observed prenatally, and their perinatal clinical features and management were compared. Prenatal findings, such as oligohydramnios, mode of delivery, indications for emergency cesarean delivery, and fetal imaging findings observed using ultrasonography or MRI, recorded in the maternal medical charts were reviewed. The diagnosis of pulmonary hypoplasia using fetal MRI was not typical for fetal ascites cases, because diaphragmatic compression caused by ascites may temporarily reduce the size of the mature lungs. Therefore, the finding of pulmonary hypoplasia was judged comprehensively by the radiologist, who considered not only the lung size but also the ratio of the fetal lung to liver signal intensity using T2-weighted imaging [9]. Postnatal findings, such as Apgar scores at 1 min and 5 min, gestational age at birth, birth weight, and serum C-reactive protein (CRP) and creatinine levels at birth, recorded in the medical charts of the fetal patients were reviewed. Because the latest diagnosis of persistent cloaca was determined immediately after birth for all patients, blood samples were collected a few hours after birth when intravenous treatment was started. The CRP and serum creatinine levels in the blood samples of newborns scheduled to undergo surgery were evaluated. The postnatal findings and medical outcomes, such as the durations of mechanical ventilation and oxygen administration, age at initial discharge, serum creatinine level at 1 year, and age at the last follow-up, and the surgical outcomes, such as the occurrence of adhesive ileus before 1 year, initial urinary drainage after birth, the need for a urinary drainage catheter at initial discharge, length of the common channel, age at the time of anorectal vaginoplasty, and initial urinary drainage procedures after birth was performed, were also reviewed. The relationship between these clinical findings and the presence of fetal ascites was analyzed. The length of the common channel was measured using endoscopy at the time of anorectal vaginoplasty or during the preoperative examination. An assessment of spontaneous voiding was performed by a pediatric urologist at birth to determine whether surgery was necessary. If surgery was required, then a catheter was placed for colostomy surgery. When the patient's general condition stabilized after surgery, spontaneous voiding was reevaluated to determine whether the catheter could be removed before discharge home. Catheterization was performed for patients who were unable to adequately void independently because of obstruction of the common channel. Some patients received vaginal catheterization to drain the refluxed urine because of difficulty placing the catheter in the bladder.

Statistical analysis

Statistical analyses were performed using JMP software (version 13.0; SAS Institute Inc., Cary, NC, USA). The statistical tests included the chi-square test and Mann–Whitney *U* test. Data are reported as the median and interquartile ranges. Statistical significance was defined as $P < 0.05$.

Ethical considerations

Approval for this study was obtained from the Institutional Review Board of Osaka Women's and Children's Hospital (protocol no. 1459). The procedures used for this study adhered to the tenets of the Declaration of Helsinki. The requirement for signed informed consent was waived because of the retrospective study design and use of de-identified data. Details of the study were published on an institutional website, and individuals had the right to decline participation.

Results

Seventeen patients were enrolled in this study during the study period (Table 1). The presence of fetal ascites was recognized in seven patients by fetal ultrasonography or fetal MRI. No fetal ascites were observed in 10 patients. A comparison of clinical parameters of patients with and without fetal ascites is shown in Table 1. Nine findings were significantly related to the presence of fetal ascites (with

Table 1 Comparison of clinical parameters of patients with persistent cloaca with and without fetal ascites

	Total	With fetal ascites	Without fetal ascites	*P* value
Fetal ascites, *n* (%)	17 (100)	7 (41)	10 (59)	–
Oligohydramnios, *n* (%)	4 (41)	2 (30)	2 (20)	0.56
Gestational age at birth (weeks)[a]	37.0 (30.6–41.0)	34.7 (30.6–37.0)	39.3 (30.6–41.0)	0.192
Preterm birth, *n* (%)	8 (47)	6 (86)	2 (20)	0.015*
Birth weight (g)[a]	2888 (1404–3524)	2888 (1566–3080)	2856 (1404–3524)	0.921
Birth weight z-score[a]	0.86 (− 3.49–4.91)	1.5 (0.21–4.91)	− 0.2 (− 3.49–2.08)	0.008*
Emergency cesarean delivery, *n* (%)	8 (47)	6 (86)	2 (20)	0.015*
Apgar score at 1 min[a]	4.5 (2–9)	3.0 (2–6)	8.0 (3–9)	0.004*
Apgar score at 5 min[a]	9 (5–10)	7.0 (5–9)	9.0 (7–10)	0.022*
Serum CRP level at birth (mg/dL)[a]	0.0 (0.00–16.25)	5.4 (0.01–16.25)	0.0 (0.00–0.08)	0.026*
Serum creatinine level at birth (mg/mL)[a]	0.61 (0.24–0.86)	0.54 (0.24–0.77)	0.61 (0.49–0.86)	0.291
Duration of mechanical ventilation (days)[a]	3 (0–29)	7.0 (0–29)	0.0 (0–13)	0.057
Duration of oxygen administration (days)[a]	3 (0–58)	8.0 (1–58)	0.5 (0–20)	0.047*
Serum creatinine level at the age of 1 year (mg/dL)[a]	0.25 (0.16–0.53)	0.25 (0.20–0.80)	0.25 (0.16–0.53)	0.564
Need for urinary drainage catheter at initial discharge from hospital, *n* (%)	8 (41)	6 (86)	2 (20)	0.015*
Common channel length (cm)[a]	2.9 (1.0–3.5)	3.0 (1.5–3.5)	2.0 (1.0–3.5)	0.241
Age at initial discharge (day)[a]	49 (19–156)	80 (49–156)	34 (19–143)	0.014*
Age at the time of anorectal vaginoplasty (months)[a]	15 (5–24)	13 (8–24)	16 (5–24)	0.705
Age at last follow-up (month)[a]	142 (44–290)	218 (44–258)	141 (76–290)	0.98

CRP C-reactive protein

[a]Median (range)

*Statistically significant

vs without fetal ascites): preterm birth (85.7 vs 20.0%; $P=0.015$); birth weight z-scores (1.5 vs − 0.2; $P=0.008$); emergency cesarean delivery (85.7 vs 20.0%; $P=0.015$); Apgar score at 1 min (3.0 vs 8.0; $P=0.004$); Apgar score at 5 min (7.0 vs 9.0; $P=0.022$); serum CRP level at birth (5.4 vs 0.0; $P=0.026$); duration of oxygen administration (8.0 vs 0.5; $P=0.047$); the need for a urinary drainage catheter at initial discharge (85.7 vs 20.0%; $P=0.015$); and age at initial discharge (46 vs 80; $P=0.014$).

Prenatal findings of the patients with fetal ascites and those without fetal ascites are summarized in Table 2. Fetal MRI was performed for seven patients, and a definite prenatal diagnosis of persistent cloaca was determined for six of them. Two patients in the group with fetal ascites were diagnosed with meconium peritonitis caused by intestinal perforation without any suspicion of persistent cloaca (patients 2 and 3). In patients 2 and 4, an intra-abdominal cyst was detected using fetal ultrasonography and revealed to be hydrocolpos, because the cyst resolved soon after birth. Emergency cesarean delivery was performed for eight patients because of exacerbation of hydronephrosis or oligohydramnios (four patients), fetal hydrops (two patients), early placental abruption (one patient), and nuchal cord with nonreassuring fetal status (one patient) (Table 2).

The initial urinary drainage procedure after birth and urinary drainage at discharge are summarized in Table 3. Patients with persistent cloaca were diagnosed immediately after resuscitation and blindly catheterized, often with vaginal placement. Spontaneous voiding was observed early after birth in two patients; their hydrocolpos was mild, so catheterization was not needed before surgery (patients 11 and 12). Seven patients experienced sufficient urinary drainage with the initial urinary drainage procedure. However, eight patients required catheterization under the guidance of a cystoscope or other invasive procedures because of insufficient urinary drainage. Four patients required invasive procedures, such as initial urinary drainage after birth (patients 2, 3, 6 and 8). Patient 8 had a very low birth weight and bilateral hydronephrosis, was unable to void adequately, and could not undergo cystoscopy; therefore, a left nephrostomy was created when a colostomy was created the day after birth. Eight patients required some type of urinary drainage procedure at discharge. Five patients needed clean intermittent catheterization of the bladder or vagina performed by the patient's family. Three patients required a different method of urinary drainage.

Only one patient with fetal ascites (patient 3) underwent transuterine ascites puncture as a fetal intervention. No

Table 2 Prenatal findings of patients with persistent cloaca with and without fetal ascites

Patient number	Gestational week at development of FA	Gestational week at fetal MRI	Definite prenatal diagnosis of PC	Prenatal imaging findings	Indications for emergency cesarean delivery
1	32	NP	No	FA, hydronephrosis, hydrometrocolpos	NP
2	20	NP	No	FA, oligohydramnios, hydronephrosis, pulmonary hypoplasia, abdominal cyst	Early placental abruption
3	24	NP	No	FA, hydronephrosis, hydrometrocolpos, abdominal cyst	Exacerbation of hydronephrosis
4	28	NP	No	FA, fetal hydrops, abdominal cyst	Fetal hydrops
5	24	24	Yes	FA, cloaca, fetal hydrops, pulmonary hypoplasia, double vagina	Fetal hydrops with mirror syndrome
6	25	29	Yes	FA, oligohydramnios, cloaca, hydronephrosis, bicornuate uterus	Exacerbation of oligohydramnios and arrested labor
7	33	29	Yes	FA, cloaca, oligohydramnios, hydronephrosis, hydrocolpos, double vagina, double uterine	Exacerbation of oligohydramnios and hydronephrosis
8	-	NP	No	Oligohydramnios, hydronephrosis, obstructive uropathy	NP
9	–	NP	No	No abnormal findings	NP
10	–	28	Yes	Oligohydramnios, right ureterohydronephrosis, left renal hypoplasia, bicornuate uterus	Exacerbation of oligohydramnios and arrested labor
11	–	NP	No	No abnormal findings	NP
12	–	36	No	Hydrocolpos	NP
13	–	NP	No	Hydronephrosis, hydrometrocolpos,	NP
14	–	36	Yes	Hydronephrosis, hydrocolpos	NP
15	–	30	Yes	Right renal agenesis, left renal cyst, hydrocolpos,	Nuchal cord, nonreassuring fetal status
16	–	NP	No	Hydrocolpos	NP
17	–	NP	No	No abnormal findings	NP

NP not performed, *MRI* magnetic resonance imaging, *PC* persistent cloaca

FA fetal ascites, *MP* meconium peritonitis

patients developed adhesive ileus before 1 year, regardless of fetal ascites development.

Discussion

To our knowledge, this is the first case–control study to evaluate the impact of prenatal fetal ascites on postnatal clinical features and management. We focused on fetal ascites with persistent cloaca, because they are associated with chemical peritonitis caused by the backflow of urine and meconium into the abdominal cavity through the fallopian tubes. The group with fetal ascites had more births performed via emergency cesarean delivery, was at higher risk for low birth weight and premature birth, and tended to have a worse general condition at resuscitation. Postnatally, the group with fetal ascites was at higher risk for a worse respiratory status, longer hospital stays, and the need for a catheter at discharge. However, all patients were discharged from the hospital and subsequently underwent anorectal vaginoplasty. The high CRP level at birth could reflect chemical peritonitis, which itself is not a lethal risk factor.

In this study cohort, seven of 17 patients were diagnosed with fetal ascites (41%), resulting in a cohort with an approximately higher proportion of patients with fetal ascites than the actual proportion of patients with fetal ascites and persistent cloaca; however, the successful diagnostic rate for fetal ascites in the overall population of patients with persistent cloaca is unknown. This discrepancy is hypothesized to have occurred, because patients with persistent cloaca and fetal ascites were considered to have a greater obstruction of the common channel, which made significant findings, such as hydrocolpos or ascites by routine fetal ultrasonography more noticeable, resulting in a higher fetal diagnosis rates. Regarding the extent to which fetal ascites are detectable by ultrasound, there were no patients with high CRP levels in

Table 3 Urinary drainage for patients with persistent cloaca with and without fetal ascites

Patient number	Fetal ascites	Initial urinary drainage procedure after birth	Urinary drainage at discharge
1	Detected	Vaginal catheterization	CIC
2	Detected	Bilateral vaginal puncture, bladder puncture	Cutaneous vesicostomy
3	Detected	Vaginal catheterization	CIC
4	Detected	Bladder catheterization	Unnecessary
5	Detected	Abdominal puncture, cutback procedure of common channel, catheterization into bladder and vagina	CIC
6	Detected	Cutaneous vaginostomy of the *left vagina, catheterization of the right vagina and bladder	Indwelling urinary catheterization
7	Detected	Catheterization of the bladder and vagina	CIC
8	ND	Left nephrostomy	Bilateral nephrostomy
9	ND	Bladder catheterization	Unnecessary
10	ND	Bladder catheterization	Unnecessary
11	ND	Unnecessary	Unnecessary
12	ND	Unnecessary	Unnecessary
13	ND	Bladder catheterization	Unnecessary
14	ND	Bladder catheterization	Unnecessary
15	ND	Bladder catheterization	Unnecessary
16	ND	Bladder catheterization	CIC
17	ND	Bladder catheterization	Unnecessary

ND not detected, *CIC* clean intermittent catheterization

*The condition of patient 6 was complicated by a double vagina

the group without fetal ascites, suggesting that fetal ultrasound would not have missed the cases involving peritonitis. In other words, patients with persistent cloaca and undetected fetal ascites did not have peritonitis at birth.

We investigated the indications for an emergency cesarean delivery, because it was significantly more common for patients with fetal ascites. The two major causes of emergency cesarean delivery associated with persistent cloaca with ascites were progressive fetal hydrops and progressive hydronephrosis with oligohydramnios. Progressive fetal hydrops may have been caused by peritonitis. When progressive hydronephrosis with oligohydramnios occurred, we opted for emergency delivery because of the potential for the rapid development of hydronephrosis and/or oligohydramnios during late pregnancy and to prevent the exacerbation of renal damage by quickly releasing the urinary obstruction to perform urinary drainage. In addition, the higher z-score for birth weight despite the higher rate of preterm birth was likely attributable to the increased weight caused by ascites and edema induced by peritonitis.

A comparison of the postnatal parameters revealed that patients with persistent cloaca and fetal ascites had lower Apgar scores, higher serum CRP levels, and a more extended period of requiring postnatal oxygen. It is quite possible that the inflammation caused by peritonitis at birth deteriorated the general condition and affected the respiratory status. Some cases of persistent cloaca with fetal ascites have been reported to involve diaphragm elevation and lung hypoplasia caused by ascites and oligohydramnios, which may result in the need for prolonged postnatal respiratory support for severe cases. Preterm birth was a possible confounding factor for deterioration of the respiratory status. Combining these factors would have contributed to the significant deterioration of the respiratory status in our cohort.

When persistent cloaca with fetal ascites occurred, initial urinary drainage procedures were performed within 24 h of birth, and bladder catheterization using a cystoscope or invasive procedures was performed under general anesthesia. The patients tended to require urinary drainage catheters at the time of the initial hospital discharge because of urinary tract obstruction. Interestingly, despite the severity of urinary tract transit problems experienced by patients with fetal ascites, fetal ascites was not a risk factor for renal dysfunction at 1 year after birth. This result could be attributable to a phenomenon known as the pressure pop-off mechanism [10]. The overflow of urine into the abdominal cavity may act as a pressure-venting mechanism that prevents an increase in intravesical pressure, thereby moderating renal damage caused by vesicoureteral reflux. Some studies have reported that urine extravasation into the abdominal cavity has a beneficial effect on renal function in the presence of urinary ascites attributable to the posterior urethral valve

[11], [12]. This pressure pop-off mechanism and adequate postnatal urinary drainage management using catheters and invasive procedures presumably prevented more significant renal damage in patients with fetal ascites at birth and postnatally. However, the evaluation of the renal function is limited, because serum creatinine levels immediately after birth are highly dependent on the serum creatinine level of the mother. Patients with persistent cloaca undergo anorectal vaginoplasty at approximately 1 year of age, and the renal and bladder functions and serum creatinine level are influenced by that surgery. Therefore, long-term, detailed observational and analytical studies involving larger populations are necessary to determine the impact of fetal ascites on renal and bladder functions.

The presence or absence of fetal ascites with persistent cloaca is crucial to the perinatal management, because the clinical picture differs among patients. In addition, the prenatal diagnosis of persistent cloaca with fetal ascites is critical, because it can predict the need for emergency cesarean delivery, poor general conditions during resuscitation, and the requirement of invasive urinary drainage procedures immediately after birth. Therefore, for cases of unexplained fetal ascites complicated by hydronephrosis or abdominal cysts, fetal MRI is recommended, because it is better than ultrasonography for diagnosing persistent cloaca [2], [13]. Bischoff et al. reported that 62% of 95 patients with persistent cloaca had major prenatal abnormal findings on ultrasonography, but that only 6% of all cases were correctly diagnosed as persistent cloaca prenatally [2]. Piero et al. emphasized the effectiveness of MRI for determining the definite diagnosis of persistent cloaca and stated that fetal MRI should be performed to obtain additional information when ultrasonography detects the signs that can indicate an underlying multiorgan malformation [14]. Regarding the appropriate timing of MRI, although our cohort was too small to properly assess the positive diagnosis rate, the group with fetal ascites was born at a median of 34.7 weeks of gestation, suggesting that MRI should be performed before that time. An accurate prenatal diagnosis of persistent cloaca using MRI would result in the appropriate counseling of parents and allow for better perinatal management of the patients.

Previous studies have reported cases of persistent cloaca with fetal ascites and poor prognoses, such as stillbirth and neonatal death; however, no fatalities occurred during our study [3–5, 7, 15, 16]. In our study, peritonitis caused by fetal ascites was also a factor that deteriorated the general condition at birth; however, it was not fatal. Some studies reported that stillbirths or neonatal deaths after preterm birth involving very low birth weights have occurred after paracentesis of the fetal abdominal cavity or bladder was performed as fetal therapy [3, 4, 7]. In our cohort, a fetal abdominal puncture was performed only for patient 3, and an emergency cesarean delivery was performed after the puncture. The effectiveness of ascites or urine drainage as fetal therapy cannot be determined from these studies; however, there might be an increased risk of preterm delivery caused by the puncture procedure. In addition, some of the cases that resulted in stillbirth were complicated by malformations of other organs, and it is possible that diseases other than cloaca may have contributed to fetal death [5, 7]. All the aforementioned studies are case reports; therefore, nothing definitive can be concluded. Because our study was a case–control study that excluded severe airway or cardiac anomalies, the clinical effect of fetal ascites on persistent cloaca could be assessed more accurately.

There were several limitations to this study. First, this study had a retrospective design, a single-center cohort, and a small sample size. Therefore, a more extensive multicenter prospective study is required to validate the results. Second, we did not discuss the effects of fetal interventions, because they were only relevant to one patient; however, existing reports have described the effectiveness of fetal urine drainage [5, 17]. Third, our cohort did not include stillbirths or abortions. Therefore, we have not been able to completely analyze the impact of fetal ascites on the fetus. Because stillbirths and abortions may not be uncommon with this disease, a comprehensive study including such cases is required.

Conclusions

The postnatal management of patients with persistent cloaca and fetal ascites differs significantly from that of patients without fetal ascites. These infants are more likely to be born prematurely or via emergency cesarean delivery, have a worse general condition at birth, require more extended periods of oxygen supplementation after birth, require longer hospital stays, and require a catheter for urinary drainage at discharge. For patients with unexplained fetal ascites complicated by hydronephrosis or abdominal cysts, which may be caused by the backflow of urine or meconium from cloaca, MRI may be helpful for determining the definite diagnosis of persistent cloaca and its perinatal management.

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Declarations

Conflict of interest The authors have no conflict of interest to declare.

Ethics approval Approval for this study was obtained from the institutional review board of Osaka Women's and Children's Hospital (protocol no. 1459). The procedures used during this study adhered to the tenets of the Declaration of Helsinki.

Consent to participate The requirement for signed informed consent was waived because ofthe retrospective study design and the use of de-identified data. Detailsof the study were published on an institutional website, and individualshad the right to decline participation.

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