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Previable preterm premature rupture of membrane at the 19th week of gestation in a pregnancy complicated by a multicystic dysplastic kidney of the fetus – a case report

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ABSTRACT

Preterm premature rupture of membranes (pPROM) before 22–24 weeks of gestation is considered previable and poses a high risk to the chances of successful delivery. Multicystic dysplastic kidney (MCDK) is the most common form of dysplasia of this organ, usually affecting 1 kidney. A prenatal suspicion of MCDK requires escalation of the clinical process to a reference center qualified and equipped for further diagnosis and treatment. We present a unique case of prenatal pPROM at 19 weeks of gestation in a pregnancy complicated by fetal MCDK as an interesting obstetric and neonatal patient story. We have not found any published evidence on the impact of coexisting fetal congenital malformations on a pregnancy affected by previable pPROM. The aim of this study is to highlight the importance of a multidisciplinary team approach in the management of previable pPROM and the difficulties of decision-making involving both health professionals and the mother. Initially, medical management focuses on the safety of the mother, and as the fetus reaches viability, the focus shifts to the high standard of care for the child. The final successful outcome and follow-up was the effect of coordinated prenatal and postnatal medical care.
Keywords: preterm labor; preterm premature rupture of membranes; surgical treatment; complications; multicystic dysplastic kidney.

INTRODUCTION

Premature rupture of membranes (PROM) is a rupture of the amniotic sac before the onset of labor [1]. A rupture of the fetal membranes before the 37th week of pregnancy – preterm premature rupture of membranes (pPROM) affects approx. 3% of pregnancies [2]. It is a major cause of preterm birth. Obstetric care depends on the stage of pregnancy and concomitant factors such as uterine contraction activity [2]. Preterm premature rupture of membranes before 22–24 weeks of gestation is considered preterm [3].

Multicystic dysplastic kidney (MCDK) is the most common form of dysplasia of this organ, usually affecting 1 kidney [4]. In case of prenatal suspicion of MCDK, the affected infant should be born in a tertiary referral center qualified for further diagnosis and life-saving treatment [5].

We present a unique case of previable pPROM at 19 weeks of gestation in a pregnancy complicated by fetal MCDK. We found no published evidence of the influence of coexisting fetal congenital malformations on a pregnancy affected by pPROM. The aim of this study is to highlight the importance of a multidisciplinary team approach in the management of previable pPROM and the difficulties of decision-making involving both health professionals and the mother. An additional review of the literature illustrates current guidelines for the management of both pPROM and MCDK.

CASE REPORT

A 1650 g (90–97 centile for expected gestational age) male newborn of the fifth pregnancy and fifth delivery was born by cesarean section at 29 weeks' gestation to a 35-year-old mother in a tertiary obstetric unit in Poland and received Apgar scores of 7/7/7 (at 1, 5, and 10 min respectively).

Prenatally, at 19 weeks of gestation, the mother presented with water breaking for 2 days until the previable pPROM was confirmed by ultrasound (US) in a local hospital in the United Kingdom, where the mother lived during the pregnancy. In addition, dilatation of the bowel loops on examination, combined with an inability to visualize the right kidney, led to a referral to a dedicated pregnancy assessment unit. The next day, at 20 weeks 0 days, US revealed oligohydramnios and right-sided MCDK (Fig. 1) with a mass effect in the abdomen.

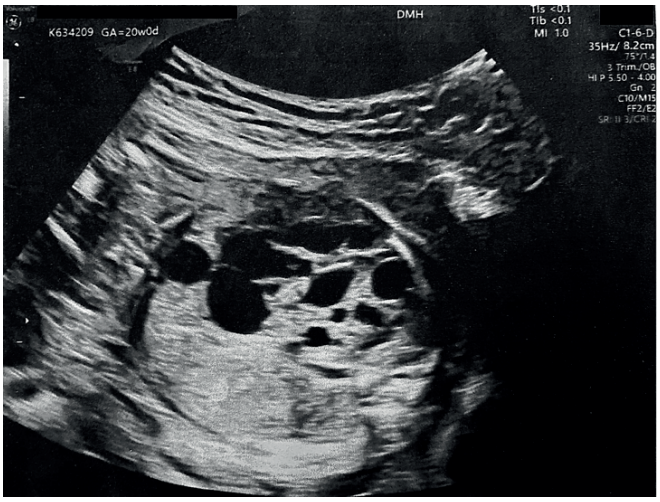

FIGURE 1. Ultrasound at 20 weeks 0 days presenting a picture of a right-sided multicystic dysplastic kidney

The mother was informed of the potential risks of continuing the pregnancy and that such a condition was an indication for abortion with her consent. Since the mother was determined to continue the pregnancy, the fetal clinical status was evaluated by US every 2 weeks and she was monitored twice a week for elevated inflammatory parameters. At 25 weeks 6 days of gestation, US revealed right-sided pulmonary hypoplasia unilateral to the renal anomaly. Due to the potential multiple complications of unilateral MCDK, magnetic resonance imaging (MRI) was scheduled at 30 weeks' gestation with multidisciplinary consultation. During pregnancy, the mother was treated twice with nitrofurantoin and penicillin for urinary tract infection (UTI).

In the 28th week of pregnancy the mother decided to come to Poland. During her hospitalization in the Department of Perinatology and Gynecology, she underwent a full course of steroid therapy.

With satisfactory cardiotocography and the absence of symptoms of intrauterine infection, there was no indication for antibiotic therapy. After 4 days, symptoms of a dynamically developing intrauterine infection were observed and the decision was made to deliver the baby. A cesarean section was indicated because of the mother's vascular brain malformation. After delivery, the newborn required positive pressure ventilation and was transferred to the Neonatal Intensive Care Unit (NICU) on noninvasive ventilation in biphasic mode. Due to respiratory failure, the boy was intubated and required mechanical ventilation for 2 days, then non-invasive support, first with biphasic positive airway pressure (until 7 days of age), then high-flow nasal cannula (until 15 days of age), and passive oxygen therapy until 47 days of age, leading to a clinical diagnosis of bronchopulmonary dysplasia (BPD). At birth, the boy was diagnosed with sepsis following a UTI. Laboratory tests at 6 h of age revealed positive inflammatory parameters (C-reactive protein – 12.21 mg/L; procalcitonin – 168 ng/mL). Microbiological cultures (blood, cerebrospinal fluid) were negative for pathogenic flora, and *Streptococcus agalactiae* was detected in an ear swab taken immediately after birth. On physical examination, a large tumor was palpable on the right side, modulating the contour of a diffuse abdomen. An X-ray showed obvious mass effects of the right MCDK and pneumonia, but no features of respiratory distress syndrome (RDS) – Figure 2.

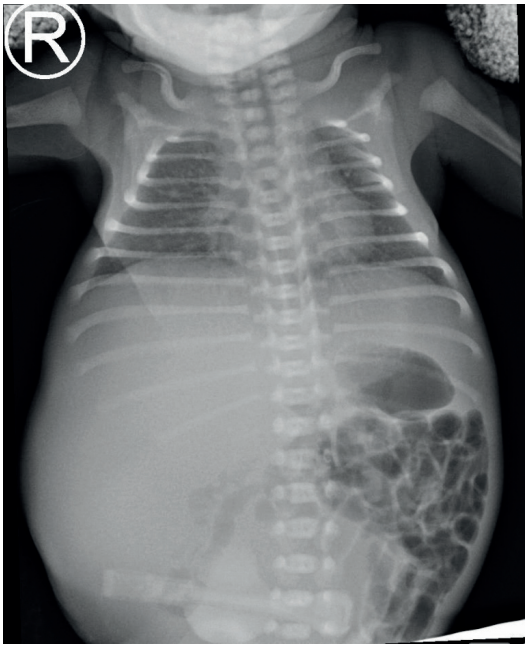

FIGURE 2. X-ray performed on the first day of life: large right-sided abdominal mass and pneumonia

Ultrasound performed on day 2 of life showed a polycystic structure in the right abdomen with its lower pole crossing the midline and reaching the lower pole of the left kidney. Laboratory parameters of renal function were elevated. Bladder catheterization confirmed operative diuresis. The newborn was referred to the neonatal clinic for further evaluation due to low birth weight and poor general condition. From the first day of life, trophic feeding with breast milk was performed, first with a probe, then with a nipple and breastfeeding. Full tolerance was achieved on the 28th day of life by discontinuing total parenteral nutrition (Numeta G13%E Preterm). Features of cerebral immaturity and increased periventricular echogenicity were noted in the brain by repeated sonograms at birth and up to 4 weeks of age. The control examination at the age of 8 weeks confirmed small periventricular cysts up to 5 mm in diameter, which were classified as second-degree bilateral leukomalacia (Fig. 3) [6].

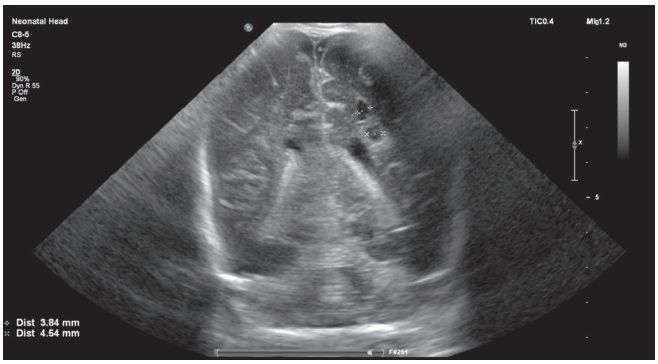

FIGURE 3. Brain ultrasound picture at 8 weeks presenting periventricular cysts under 5 mm in diameter as a sign of second-degree leukomalacia

Echocardiography showed pulmonary valve stenosis, which required further outpatient cardiological monitoring. Laboratory markers of cholestasis were observed in the fourth week of life. Toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus, and other viral infections, alpha-1-antitrypsin deficiency and metabolic disorders were excluded. Ursodeoxycholic acid was used for treatment. Because of anemia, he received 3 group-compatible irradiated leukoreduced packed red blood cell transfusions. The patient required vitamin D3 supplementation, and a sodium-phosphate solution was used for hyperparathyroidism and calcium-phosphate metabolic disorder. Iron supplementation was started at 7 weeks of age. Two ophthalmological examinations revealed no retinopathy of prematurity (ROP). At 7 weeks of age (36 weeks corrected age), with a birth weight of 2765 g (25–50 centile for expected gestational age), the boy was referred to the referral pediatric department for further diagnosis of MCDK. In the dimercaptosuccinic acid (DMSA) renal scintigraphy, the left kidney correctly accumulated the marker, while the right kidney projection showed a large photopenic area modeling the liver without marker accumulation. Proteinuria and leukocyturia were observed during hospitalization. *Klebsiella oxytoca* and *Enterococcus faecalis* were found in the urine culture. The boy received albumin infusions for hypoalbuminemia and hypoproteinemia. In the US, the right kidney filled the right abdomen, causing the mass effect, with cysts up to 4 cm in diameter without evidence of parenchyma (Fig. 4).

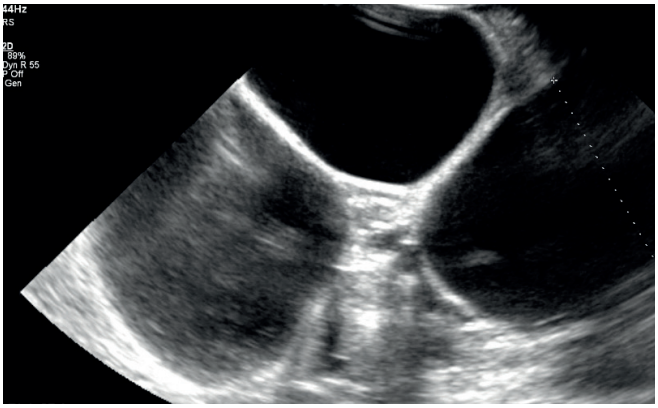

FIGURE 4. Ultrasound picture of the right kidney prior to the nephrectomy, cysts up to 4 cm in diameter with no parenchyma

On the 61st day of life, a laparotomy was performed with a horizontal approach in the right mesogastrium. When the peritoneum was opened, it was seen that the retroperitoneal space was constricted by the prominent cysts of the right kidney. A large cyst was punctured and decompressed with a needle attached to a suction device, evacuating approx. 50 mL of clear fluid content. Other cysts were then bluntly dissected and a total of approx. 300 mL was aspirated. The kidney was removed from the retroperitoneal space (Fig. 5). The ureter was not visualized and the vessels were atrophic. The postoperative course was uneventful and the patient was discharged on the fourth postoperative day. Histopathological examination of a 7 x 5 x 2 cm cross-section of the kidney revealed a severely disorganized architecture with no clear cortex or medulla and a decreased number of proximal nephron elements. The renal parenchyma was replaced by areas of fibrosis with multiple cysts, immature tubules surrounded by collarettes of smooth muscle, and foci of hyaline cartilage. These features are consistent with the diagnosis of MCDK (Fig. 6). During the subsequent outpatient follow-up, the child qualified for left-sided inguinal hernia surgery. The operation was performed in the 12th week of life without complications.

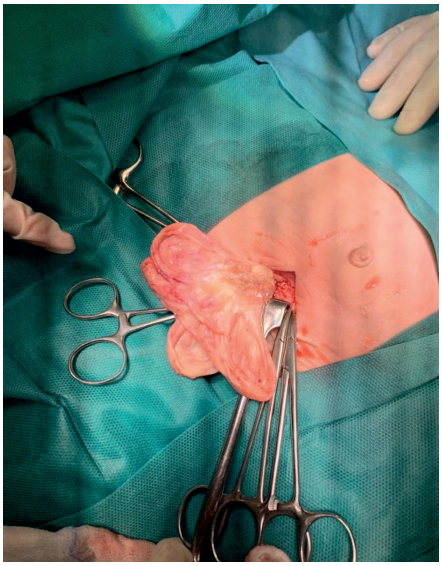

FIGURE 5. Intraoperative picture during nephrectomy

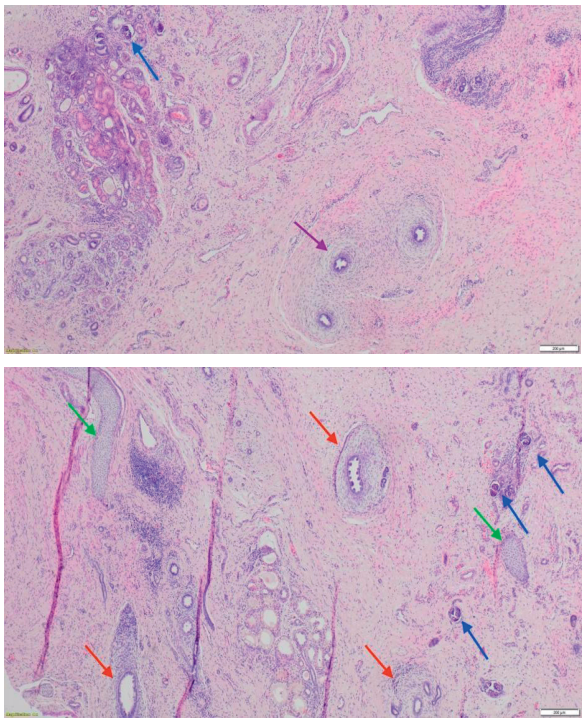

FIGURE 6. Hematoxylin and eosin staining (original magnification ×4): A – atrophic and disorganized proximal nephron elements with immature glomeruli (blue arrow), immature tubules surrounded by smooth muscle collarettes (purple arrow); B – atrophic and disorganized proximal nephron elements with immature glomeruli (blue arrow), immature tubules surrounded by smooth muscle collarettes (red arrow), foci of hyaline cartilage (green arrow)

Currently, the boy is 11 months old and in good general condition. He remains under outpatient neurological control with an intensive rehabilitation protocol including the Bobath method with mild motor deficits (e.g. upper limb support). The boy has no diagnosed visual deficits. He continues to receive multidisciplinary care, including a nephrologist (no complications, good single kidney function) and a cardiologist (no indication for surgical intervention for pulmonary stenosis).

DISCUSSION

Premature rupture of membranes may result from complex and multifaceted pathways that contribute to the weakening of membrane morphology through alteration of the collagen network and/or activation of matrix metalloproteinases triggered by bacterial products or pro-inflammatory cytokines associated with intrauterine infection [2].

There are many known risk factors for pPROM, including low socioeconomic status, maternal nicotinism, the presence of sexually transmitted diseases, and factors that cause excessive stretching of the uterine muscle, such as polyhydramnios, multiple pregnancies, abnormal fetal positioning, and patients with a history of preterm labor [7].

The literature shows that up to half of patients with pPROM have positive amniotic fluid cultures [2]. The risk of developing an intrauterine infection increases with time after the detection of the rupture of membranes [8].

In the present case, symptoms of rupture were observed at 19 weeks' gestation, fulfilling the definition of previable pPROM [3]. There are no clear guidelines for the clinical management of such an early event. LeMoine et al. studied previable pPROM between 18–22 weeks and the survival rate was only 28%. However, antibiotherapy was associated with prolongation of pregnancy in the study [9].

According to various sources, a delivery is considered a miscarriage if it occurs before 22–24 weeks of gestation [10]. Premature rupture of membranes at this time is called previable pPROM, as the chances of survival at delivery are minimal. Based on the "Recommendations for the management of the mother and the newborn at the edge of survival": in the case of extreme prematurity (gestational age 22–23 weeks), active resuscitation is considered after birth, depending on the presence of factors improving the prognosis (morphological maturity greater than expected and birth weight, female sex, cord clamping, spontaneous motor activity, prenatal steroid therapy, intrapartum tocolysis) or at the explicit request of the parents [11].

Gestational age at delivery has a major impact on neonatal survival prognosis. Expectant management improves neonatal survival by approx. 2% for each additional day of *in utero* maturation, with optimal benefit between 24–27 weeks [2]. The literature shows that the latency period between rupture of membranes and birth is negatively related to gestational age and is typically 3 days. Supplemental antibiotic therapy prolongs it to only 6 days [7, 12].

In the case of pPROM associated with sepsis, termination of pregnancy is recommended regardless of gestational age, as it is associated with higher maternal morbidity [13]. In this case, the pPROM occurred in the 19th week of pregnancy, when there was no chance for the fetus to survive. The mother received this information at a very early stage of pregnancy but decided to continue the pregnancy. However, the latency period was 10 weeks. After initial antibiotic therapy in a gynecological ward in the UK, the patient was discharged home. During this time, the mother was under continuous outpatient care and the fetus was diagnosed with MCDK. During follow-up, the patient was treated twice with outpatient antibiotics for UTI.

Early detection of intrauterine infection is of paramount importance in monitoring the latency period of pPROM. Research shows that most infections are subclinical [2]. According to current UK guidelines (Guidelines of the Royal College of Obstetrics and Gynaecology – RCOG), routine monitoring includes maternal temperature, heart rate, blood pressure, and a combination of blood tests, as well as fetal heart rate [14].

Researchers have questioned the usefulness of individual laboratory tests that are evaluated separately (leukocytosis, CRP). A full combination of these tests should be used to diagnose infection in women with pPROM [15]. Microbiological examination of cervical smears in the diagnosis of early intra-amniotic infection [16] should also influence the choice of antibiotic prophylaxis [17]. The literature shows that the absence of fetal tachycardia basically excludes infection within the fetal membranes [18].

In the analyzed case, a wait-and-see approach was adopted, based on twice-weekly observation of symptoms of intrauterine infection (monitoring of inflammatory parameters) to reduce the risk of maternal sepsis. This procedure was partially in accordance with the guidelines of the Polish Society of Gynecologists and Perinatologists (PTGiP), which recommends expectant management with steroid therapy and prophylactic antibiotic therapy in the case of pPROM before the 34th week of pregnancy, in the absence of symptoms of intra-amniotic infection, in order to reduce the risk of intra-amniotic infection and prolong the duration of pregnancy [19].

The use of antibiotics shortly after the onset of pPROM significantly reduces the number of deliveries within the next 48 h, the occurrence of serious infection in the newborn, and contributes to the reduction in the number of serious complications (death, RDS, early sepsis, intraventricular hemorrhage, necrotizing enterocolitis) [20, 21]. After diagnosis of pPROM, the mother was monitored, including 4 fetal US before admission to the obstetric unit. During these visits, the focus was on the fetus. The assessment of fetal biometry and anatomical anomalies were combined with a Doppler test. Fetal heart rate was measured without cardiotocography until admission at 29 weeks according to NICE guidelines [22].

The incidence of severe complications and neonatal mortality due to pPROM is very high [23]. In an analysis of women with pPROM at less than 24 weeks' gestation, Chauleur et al. found that half of the newborns died despite treatment [10]. Prenatal steroid therapy reduces the incidence of RDS, intraventricular

hemorrhage, and necrotizing enterocolitis in the newborn [24], but recommendations for its use differ between the RCOG and the PTGiP.

According to PTGiP, in the absence of maternal symptoms of infection, a 48-hour cycle of steroid therapy is recommended between 24–34 weeks of gestation. It is not recommended to repeat the cycle after delivery within 7 days. In the case discussed, no steroid therapy was administered during the stay in the UK due to the initial significant immaturity of the fetus at the time of pPROM. The RCOG recommends the administration of corticosteroids for pPROM between 24–35 + 6 weeks of gestation [14]. We did not find any information in the medical literature regarding the use of corticosteroids between 21–27 weeks of pregnancy.

In the 28th week of pregnancy, the mother decided to come to Poland. During her hospitalization in the referral department of perinatology and gynecology, she underwent a full course of steroid therapy. In the absence of symptoms of intrauterine infection, there was no indication for antibiotic therapy. After 4 days of hospitalization, symptoms of a dynamically developing intrauterine infection were detected, and it was decided to terminate the pregnancy by cesarean section in the 29th week of gestation.

In the presented case study, unilateral MCDK of the right kidney was diagnosed at the time of US examination, when a previous pPROM was confirmed. Multicystic dysplastic kidney is characterized by an enlarged kidney with the presence of cysts of various sizes and the absence of normal renal parenchyma [25, 26]. Consistently diagnosed clusters of multicystic dysplasia have historically been classified as Potter type II [27, 28]. Multicystic dysplastic kidney is the most common form of renal dysplasia, usually affecting 1 kidney. It occurs at an incidence of 4.12 : 10,000, with a live birth prevalence of 3.06 : 10,000 [29]. In most cases it is sporadic, although it can run in families [5].

In the constantly updated classifications, MCKD is in the nonheritable group compared to the well-known heritable autosomal recessive polycystic kidney disease (ARPKD) and autosomal dominant polycystic kidney disease (ADPKD) associated with the polycystic kidney and liver disease 1 *(PKHD1)* gene and polycystic kidney disease (PKD) – *PKD1* or *PKD2* genes, respectively [28]. Although these mutations are not associated with MCDK, a recently published study using array comparative genomic hybridization shows that cytogenetic aberrations sometimes present in patients with MCDK may include *PKD1P1* – a pseudogene of unknown role that shares up to 97.7% sequence identity with the *PKD1* gene [30].

In most cases of MCDK, the deformed kidney is not functional and is associated with an increased risk of lower urinary tract and contralateral renal anomalies up to 75% [31]. Multicystic dysplastic kidney is commonly diagnosed in the US, but Ji and Dong show its inaccuracy and suggest additional fetal MRI screening, which is not only more accurate but can provide additional information for the assessment of MCDK [32].

Magnetic resonance imaging can differentiate between MCDK and other cystic kidney diseases, as well as identify both coexisting urinary tract malformations and non-renal anomalies such as pulmonary hypoplasia. These findings may play a role in prenatal counseling. In cases of oligohydramnios, US cannot accurately diagnose bilateral renal disease, which is correlated with higher neonatal mortality or need for dialysis in bilateral MCDK [33].

In our case, MRI was considered and scheduled for the 30th week of pregnancy, but the mother of the child changed her residence and the child was born at the 29th week of pregnancy. If the initial diagnosis is made prenatally, a voiding cystourethrography and isotope scan should be performed a few days after birth to determine renal function and detect any previously hidden abnormalities [31]. In our case, DMSA confirmed the complete lack of function of the right kidney.

The detection rate of MCDK at the prenatal stage is high, with cysts visible in the US at 15–20 weeks of gestation [33]. The recommended management of MCDK is US control, nephrological care, and assessment of laboratory markers of renal function. The most common is involution of the lesion, and the risk of neoplasia is similar to the population risk [5]. Indications for nephrectomy include the presence of cysts of enormous size, enlarging or non-involving during observation [31].

In the presented case, due to significant immaturity, low birth weight, and unstable general condition after birth, the diagnostic procedures were limited to US examinations and laboratory markers of renal efficiency. Multiple US scans confirmed the presence of a large structure in the kidney, which required surgical removal after no right-sided renal function was seen in DMSA. Histopathological examination of the removed organ was consistent with the diagnostic criteria of MCDK [34].

The clinical picture of our patient included severe respiratory failure. The boy required prolonged oxygen supplementation, which fulfilled the criteria for BPD. According to the current guidelines of the National Institute of Child Health and Human Development (NICHD), the condition is recognized when oxygen supplementation is required for at least 28 days postnatal age or 36 weeks postmenstrual age [35]. Despite the classical sequence of prematurity and the precipitating, protective and modulating factors in the pathogenesis of BPD, the right-sided pulmonary hypoplasia detected at 25 weeks of gestation, despite a normal appearance at 20 weeks of gestation, is noteworthy. We can assume that the mass effect of the enlargement of the MCDK on the same side affected the lung, in agreement with a case presented by Pettit and Chalmers [36].

The child's survival and uneventful follow-up are optimistic. Nevertheless, the need for a multidisciplinary approach both prenatally and postnatally in the described case should be emphasized. The clinical interventions during pregnancy and after birth were aimed at increasing the chances of survival of the child. This story has an amazingly good clinical long-term follow-up with a statistical race regarding the early onset of previable pPROM and multiple congenital malformations. However, well-established guidelines were implemented in this challenging situation with a successful transition to the neonatal period.

Based on the current literature, the very first step in the confirmation of previable pPROM is counseling regarding the pros and cons of continuing the pregnancy [2]. In our case, the addition of a major congenital malformation that posed an additional risk to the overall outcome, with the mother determined to continue the pregnancy, escalated the clinical approach to ethical and management challenges.

As a result of our scientific research, we conclude that the current clear guidelines for the management of pPROM provide the obstetrician with a clear roadmap to guide a patient whose child is further challenged with a congenital anomaly. Our patient was monitored regularly with an emphasis on screening for maternal infection to limit the risk of sepsis. However, there were four additional obstetric consultations with ultrasound, and detailed analysis of management. An MRI was planned, as well as referral to a high-level center for further nephrological and surgical management.

CONCLUSIONS

Our case of previable pPROM at 19 weeks' gestation in a pregnancy complicated by fetal MCDK illustrates a wide range of multidisciplinary clinical efforts to support the chances of a good pregnancy outcome despite indications to consider termination. The mother's decision to continue such a high-risk pregnancy was met with nonjudgmental support from physicians. Antenatal care was focused on the safety of the mother and included preparation for the delivery of a premature newborn with the need to expand the diagnostics and treatment of renal pathology. Postnatal care combined intensive neonatal care with interdisciplinary consultations and later surgical intervention. The child requires intensive rehabilitation, but a good general condition and optimistic psychomotor development are the basis for a positive final evaluation.

We conclude that previable pPROM individually represents such an extreme risk of pregnancy failure that the additional congenital anomaly of unilateral MCDK does not alter early obstetric care, but requires enhanced diagnosis and management once the fetus survives to a viable stage. The current state of obstetric and neonatal care provides the tools for physicians to be prepared to support such extremely challenging pregnancies.

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