International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Shekhawat D et al. Int J Reprod Contracept Obstet Gynecol. 2026 Jun;15(6):2259-2262
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20261644

Case Report

Maternal congenital renal tract anomaly as a possible risk factor for preterm prelabor rupture of membranes and placental abruption: a rare case of contracted kidney with duplex ureter

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Received: 08 April 2026
Accepted: 08 May 2026

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ABSTRACT

Congenital renal tract anomalies are uncommon in pregnancy but may predispose to adverse obstetric outcomes. We report the case of a 26-year-old primigravida with a contracted kidney and duplex ureter who developed preterm prelabor rupture of membranes (PPROM), followed by placental abruption at 32 weeks of gestation. Chronic structural renal abnormalities may contribute to recurrent infections and persistent inflammation, leading to weakening of foetal membranes and vascular compromise. This case highlights a possible pathophysiological association between congenital renal anomalies, PPROM and placental abruption. Early recognition and multidisciplinary management are essential to optimize maternal and fetal outcomes.

Keywords: Duplex ureter, Contracted kidney, PPROM, Placental abruption, Renal anomaly

INTRODUCTION

Congenital anomalies of the urinary tract, such as duplex ureter, occur in approximately 0.8-1% of the population and are often associated with urinary stasis and recurrent infections, as described by Privett et al.[1] Although frequently asymptomatic, these anomalies may gain clinical significance during pregnancy.

Renal disease in pregnancy has been associated with adverse outcomes including preterm labor, PPROM, and placental complications.[2] Infection and inflammation are major contributors to the development of PPROM, while vascular dysfunction is implicated in placental abruption.[3,4] However, the relationship between congenital renal anomalies and these obstetric complications remains underreported.

CASE REPORT

A 32-week primigravida was referred to our institute with complaints of leaking per vaginum and abdominal pain of 6-hour duration. At the referring centre, she had received a dose of antenatal corticosteroid (injection betamethasone) at 1:00 AM and was started on Alamine infusion. On admission, she was hemodynamically stable. Per abdominal examination revealed a uterus corresponding to 32 weeks' gestation, relaxed in tone, with a cephalic presentation, and fetal heart rate localized along the left Spino umbilical line. A provisional diagnosis of prelabor preterm rupture of membranes was made, and the patient was admitted for expectant management with close maternal and fetal surveillance. After 12 hours of admission, she developed worsening abdominal pain with persistent leaking per vaginum. On reassessment, her vital parameters remained stable; however, abdominal examination revealed a tense and tender uterus of 32 weeks size with cephalic presentation, and fetal heart rate was noted to be 80 beats per minute. Cardiotocography demonstrated a pathological trace characterized by persistent fetal bradycardia for 30 minutes, consistent with acute fetal compromise as per NICE guidelines. In view of the pathological CTG findings, an emergency lower segment caesarean section was performed. Intraoperative

findings confirmed the presence of placental abruption. Patient revealed history of dysuria one year back, for which she did urine routine and microscopic suggestive of 6-8 pus cells, rest normal. Urologist suggested ultrasound KUB which suggested right sided duplex ureter with right contracted kidney along with hydronephrosis. She underwent DTPA scan (Figure 3).

Management

Management was guided by recommendations from the American College of Obstetricians and Gynaecologists: Intravenous antibiotics, continuous foetal monitoring, Inj. Betamethasone 12 mg IM given at 1 AM already from the referral centre. Due to pathological CTG (acute bradycardia) and suspected placental abruption, an emergency caesarean section was performed.

Outcome

Early preterm neonate weighing 2.1 kg delivered and admitted to NICU. Placental examination revealed retroplacental hematoma (Figure 2) confirming abruption mother had an uneventful postoperative recovery.

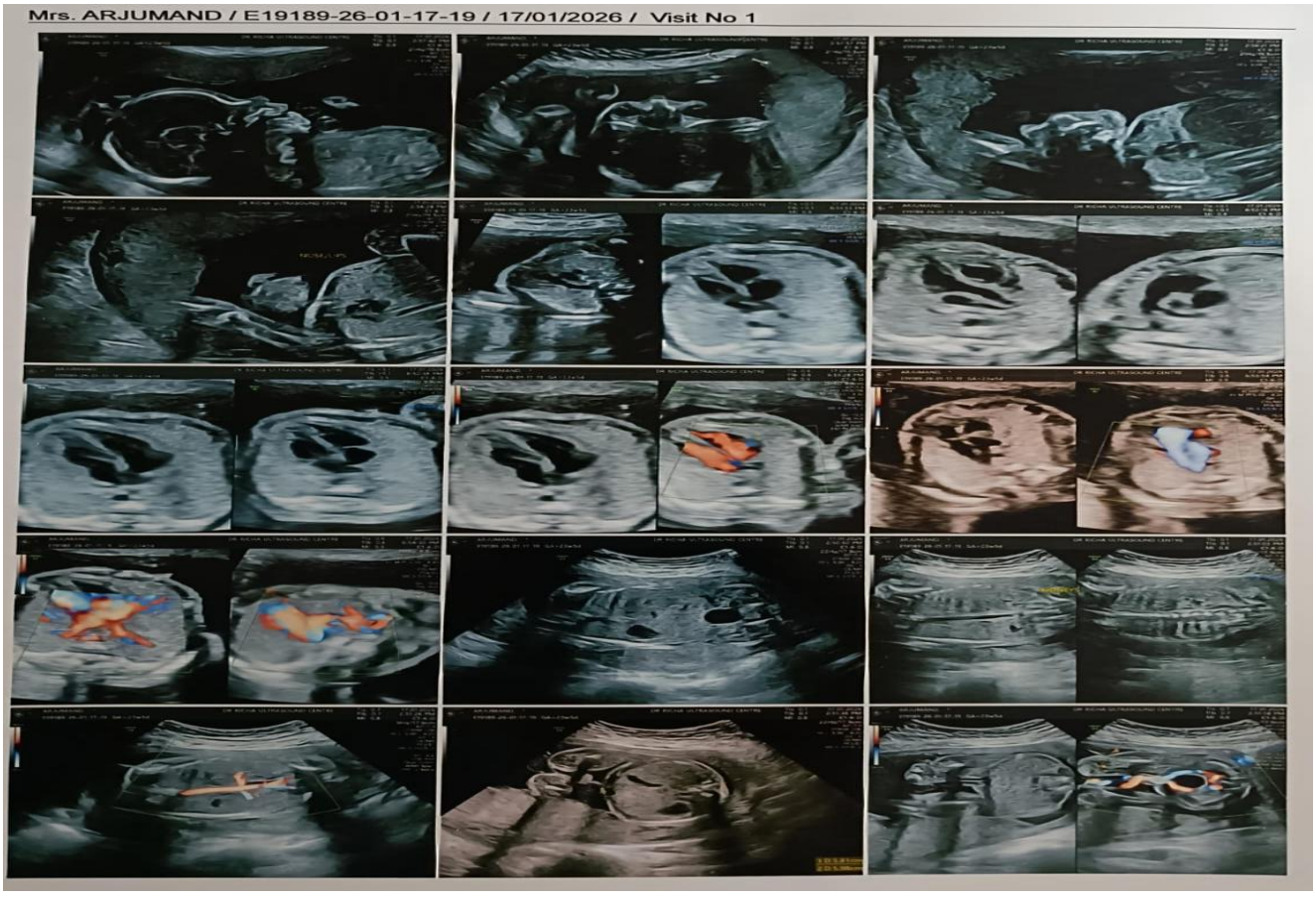

Figure 1: Ultrasound finding showing (GCMF scan).

*23 weeks 5 days gestational age, placenta-posterior, presentation-variable, amniotic fluid-adequate.

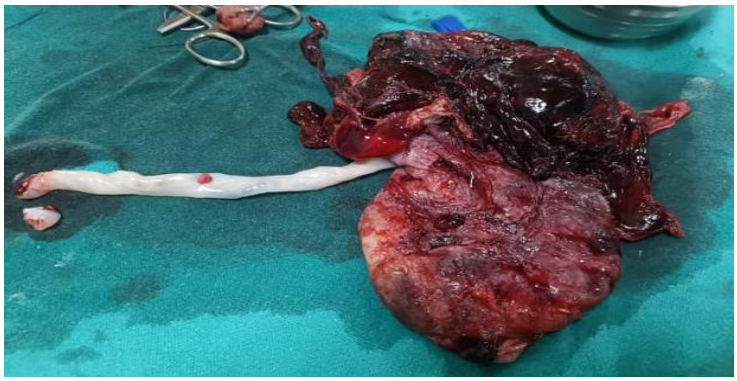

Figure 2: Intraoperative placenta showing retroplacental clot/abruption.

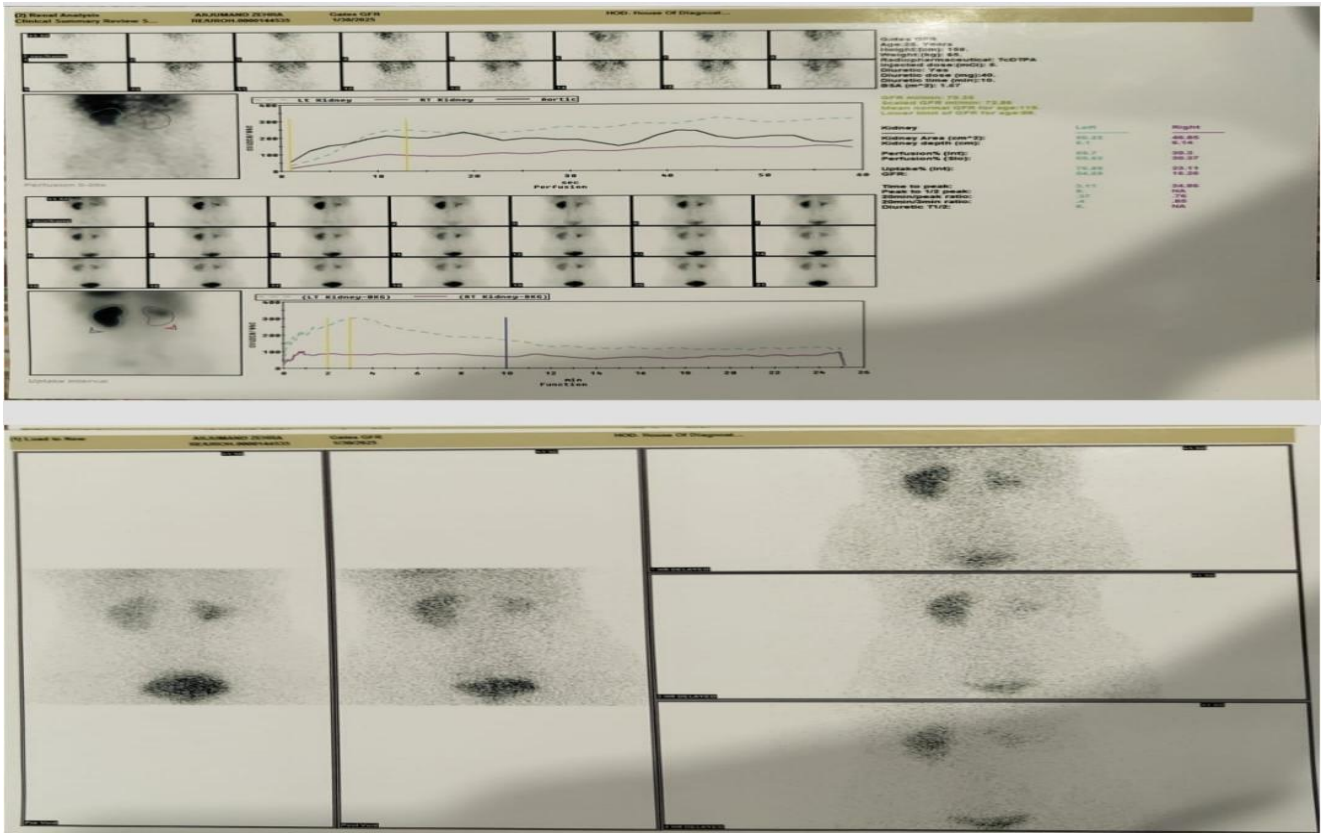

Figure 3: Diethylenetriaminepentaacetic acid (DTPA renal scan).

DISCUSSION

This case highlights a possible association between congenital renal tract anomalies and adverse pregnancy outcomes, particularly PPROM and placental abruption.

Renal anomalies and PPROM

Congenital anomalies such as duplex ureter predispose to urinary stasis and recurrent infections. These infections are strongly associated with PPROM through inflammatory mechanisms involving cytokine release and membrane weakening.

Zhou et al demonstrated that urological disorders in pregnancy are associated with an increased risk of adverse outcomes, including PPROM.[5] Similarly, Machura et al reported higher rates of infection-related complications in pregnancies complicated by renal pathology.[2] Clinical guidelines also emphasize infection as a major etiological factor in PPROM.[3]

Renal disease and placental abruption

Placental abruption is associated with vascular dysfunction, inflammation, and placental hypoperfusion. Chronic renal disease contributes to endothelial dysfunction and systemic inflammation, thereby increasing susceptibility to placental complications.

Piscitani et al highlighted the role of renal disease in promoting vascular dysregulation during pregnancy.[6] Additionally, PPROM itself may increase the risk of placental abruption due to uterine decompression and altered placental attachment.[4]

Proposed pathophysiological link

The sequence observed in this case may be explained as follows: Congenital renal anomaly, recurrent urinary tract infections, chronic inflammation, membrane weakening, PPROM, uterine decompression and vascular compromise and placental abruption.

Clinical implications

Congenital renal anomalies should be considered potential high-risk factors. Routine screening and prompt treatment of urinary infections are essential. Close antenatal surveillance may help reduce adverse outcomes.

This case highlights a possible association between congenital renal tract anomalies and adverse pregnancy outcomes, particularly PPROM and placental abruption.

CONCLUSION

Maternal congenital renal anomalies, such as a contracted kidney with duplex ureter, may act as indirect risk factors for PPROM and placental abruption through infectious

and vascular mechanisms. Increased awareness, early diagnosis, and multidisciplinary care are crucial to improving maternal and neonatal outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Shekhawat D, Pareek H, Kaur S, Kaur M, Singh P. Maternal congenital renal tract anomaly as a possible risk factor for preterm prelabor rupture of membranes and placental abruption: a rare case of contracted kidney with duplex ureter. Int J Reprod Contracept Obstet Gynecol 2026;15:2259-62.