International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Hussain TZ et al. Int J Reprod Contracept Obstet Gynecol. 2025 Oct;14(10):3538-3541
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20253105

Case Report

A case of late-onset twin-twin transfusion syndrome with severe fetal anemia and maternal acute kidney injury

Tasnim Z. Hussain[1]*, Mustafa Ahmed[2]

[1]Maternity Department, Basildon University Hospital, Basildon, United Kingdom
[2]Fetal Maternal Unit, Basildon University Hospital, Basildon, United Kingdom

Received: 27 July 2025
Accepted: 30 August 2025

***Correspondence:**
Dr. Tasnim Z. Hussain,
E-mail: misstahir@doctor.com

ABSTRACT

Twin-twin transfusion syndrome (TTTS) is a rare but serious complication of monochorionic twin gestation which forewarns obstetricians about the best management approach. The majority of cases are diagnosed early and managed with laser coagulation, resulting in reduced neonatal mortality. We have managed a late-diagnosis of TTTS Quintero stage 1, who was delivered due to suspicion of severe fetal anemia in the donor and cardiac overload in the recipient twin. The mother developed acute kidney injury in the early third trimester which worsened despite appropriate management. The cause of kidney injury in a mother carrying monochorionic diamniotic (MCDA) twins is unknown, yet restoration of renal function post-delivery suggests a connection. We are presenting this case as a rare presentation of severe fetal anemia in TTTS. This case demands additional research to identify the triggering factors, knowledge of which is expected to reformulate the management of MCDA twins.

Keywords: Twin-twin transfusion syndrome, Acute kidney injury, Laser coagulation

INTRODUCTION

Twin-twin transfusion syndrome (TTTS) is a well-known complication of MCDA twin pregnancy with around 15% incidence.[1] Another recognized complication of MCDA twin gestation is spontaneous twin anemia-polycythemia sequence, which is commonly noted after 26 weeks of pregnancy.[2] An ultrasound staging system, known as the Quintero classification, has been in use since 1999 for the grading of TTTS.[3] TTTS presenting before 18 weeks in one fourth of MCDA pregnancies poses a diagnostic challenge because Quintero staging cannot be used before 18 weeks.[4] The outcome of TTTS according to Quintero stages is based on the gestational age at the time of diagnosis.[5] A perinatal survival of 86% has been reported, therefore most pregnancies can be managed using a conservative approach.[1] Fetoscopic laser coagulation is a management option for pregnancies complicated by TTTS. It was initially offered between 16- and 24-week gestation. However, it can now be done at gestations earlier up to 15 weeks and late till 28 weeks, according to a new consensus.[6] Late-onset TTTS after 26 weeks is rare and raises an ethical dilemma about optimal management, since laser coagulation is generally not recommended after 26 weeks.[7]

The bulk of the literature related to twin gestations has focused around neonatal consequences for both twins. We are presenting a very interesting and rare situation of severe fetal anemia in the donor and cardiac overload in the recipient twin. Further, there was acute kidney injury in the mother.

CASE REPORT

A 44-years-old, primi gravida, who had conceived twins by in-vitro fertilization with her own eggs and donor sperm. She presented to our hospital at 10^{+2} weeks for

booking. The patient had no medical or surgical illness. She was allergic to penicillin and clindamycin. Her booking weight was 70 kg with a body mass index of 25.1. On venous thromboembolic risk assessment, she was at moderate risk for VTE. The patient was already on progesterone injections. Further, in view of the high pre-eclampsia risk, she was advised to take aspirin from 12 weeks gestation.

Her booking bloods returned normal, except for mild anemia. An ultrasound at 12^{+5} weeks gestation showed MCDA twins, who were both viable. The patient was seen by the fetal medicine unit consultant and delivery was planned between 36-36^{+6} weeks gestation, as per standard protocol for MCDA twin gestations. She was started on oral iron tablets. Fetal medicine unit protocol for twin gestations follow-up was initiated.

The patient had fetal medicine ultrasound scans from 16 to 25 weeks gestations. A growth discordance between 3.3 to 7% was noted. Fetal growth, amniotic fluid and Dopplers were normal for both twins. There was no evidence of TTTS, twin anemia-polycythemia sequence (TAP), twin reversed arterial perfusion sequence (TRAP) or selective fetal growth restriction. Cervical length measurement was normal. At 27^{+4} weeks gestation, mild polyhydramnios was noted in twin 2. The amniotic fluid index was 8.1cm. Fetal Dopplers were normal, and bladders were seen for both twins.

The patient presented to obstetric triage at 28 weeks gestation with reduced fetal movements for twin 1. She also had high blood pressure and visual disturbances. On assessment, her blood pressure was normal. But there was +2 proteinuria. She was given pre-eclampsia work-up and a weekly midwife follow-up, for blood pressure and urine dipstick check.

The following week, however, at 29^{+1} weeks gestation, the patient was admitted to hospital due to suspicion of pyelonephritis, for investigations and management. She had taken a course of oral antibiotics a week ago due to a urine infection. She had presented with abdominal pain, cramps and reduced fetal movements for twin 2. She had also complained of vaginal bleeding. The cervix was closed on speculum examination and no active bleed was demonstrated. Her urine culture was negative and renal ultrasound showed clinical signs of pyelonephritis. She was started on intravenous antibiotics. The patient's renal function after admission showed stage 1 acute kidney injury with creatinine of 110 umol/L. An AKI alert was initiated, and the patient was referred to the acute kidney injury nurses. The next day, the patient had oliguria with reduced urine output of 10-15 ml/hr. She started vomiting too. She was referred to an anesthetist and a microbiologist. She was given an additional 3 doses of intravenous gentamycin. On the following day, the patient developed shortness of breath. She had pitting edema extending to her knees. Her JVP was raised. Nephrology and an intensive care team were involved in her care. The echocardiogram showed normal ventricular function with adequate ejection fraction. There was no valvular lesion but a thin rim of pericardial effusion, 0.7cm, was noted. There was no evidence of cardiac tamponade. The patient's acute kidney injury worsened to stage 3 with creatinine levels increasing to 223 umol/L. Urea was raised at 14.4 mmol/L. No cause of her worsening renal function could be identified.

On the other hand, her fetal medicine ultrasound after admission, showed Qunitero-1 TTTS. There was 10% growth discordance in both twins, with twin 1 weighing smaller. Oligohydramnios was noted in twin 1 (AFI 3.6 cm) and polyhydramnios in twin 2 (AFI 9.1 cm). Both fetal bladders were seen. Cervix was 14 mm long, with no funneling. She was given steroids for fetal lung maturity and Magnesium sulfate for fetal neuroprotection. Her case was discussed with a tertiary care fetal medicine center, and they advised expectant management.

On the follow-up ultrasound, the middle cerebral artery Vmax for twin 1 was above 1.5 MoM, which raised a suspicion of anemia. Twin 2 had low umbilical artery PI and cardiac pulse Doppler which raised a suspicion of cardiac overload. In view of these findings, a decision for urgent delivery was made by the fetal medicine consultant. An uneventful cesarean delivery was done at 29^{+6}weeks gestation. The outcome was as follows:

Twin 1: (Donor) baby girl, birth weight 1.35 kg

Twin 1 was born with congenital anemia. Her hemoglobin was 69 g/L, and she was transfused with 2 units of blood. Her hemoglobin improved to 133 g/L after transfusion. She was intubated and treated with surfactant due to respiratory distress syndrome. She was given intravenous antibiotics on suspicion of sepsis. However, her blood culture returned negative. The cranial ultrasound was normal too. The second day after birth, she was noted to have raised bilirubin with no clinical jaundice. She was treated with phototherapy for 4 days. She further developed metabolic acidosis, which was managed by sodium bicarbonate. She was kept in the ICU for 12 days, then stepped down to a higher dependency unit. Her condition improved clinically.

Twin 2: (Recipient) baby girl, birth weight 1.465 kg.

Twin 2 was noted to have respiratory distress at birth with grunting and gasping. She was ventilated for 2 days, and was given surfactant treatment. She was given IV antibiotics for 6 days due to suspicion of sepsis. Her cranial ultrasound was normal. She was treated with phototherapy for 3 days due to raised bilirubin. She increased in weight gradually. Her observations and the blood gases remained normal.

Both twins were discharged to home, 3 weeks after delivery. The mother's urine output improved. Her blood picture of acute kidney injury reversed to completely

normal. She was discharged home on the 6th day after delivery in a stable condition.

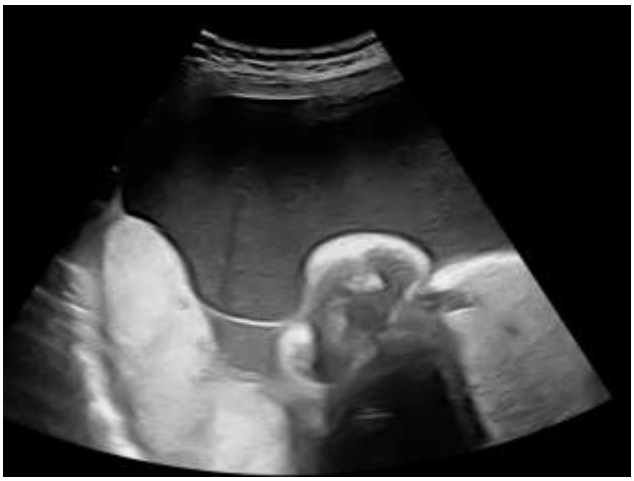

Figure 1: Oligohydramnios for twin 1 and polyhydramnios for twin 2.

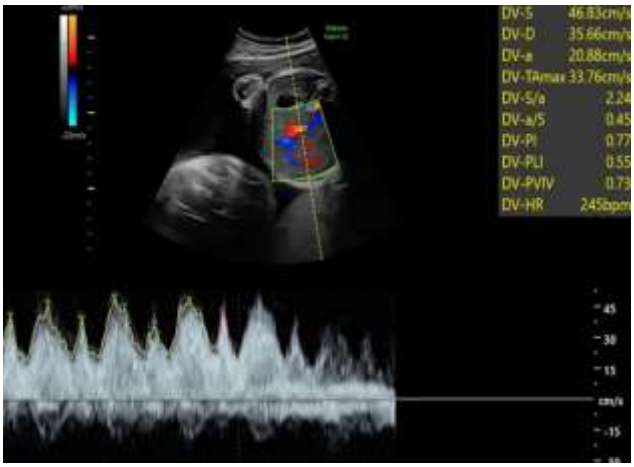

Figure 2: Ductus venosus Doppler for twin 1.

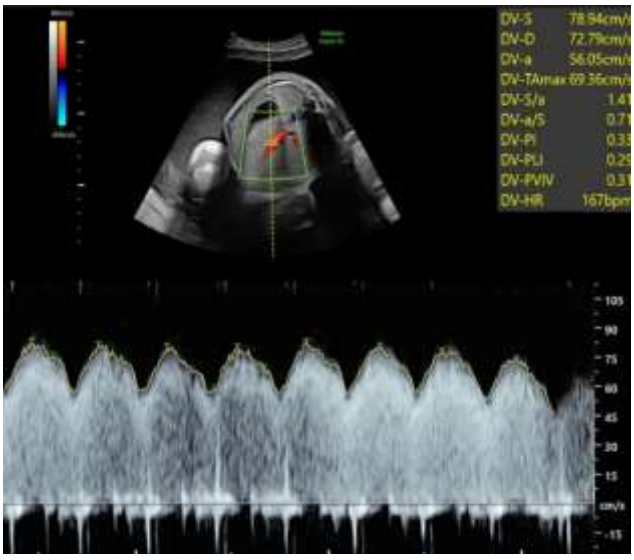

Figure 3: Ductus venosus Doppler for twin 2.

Figure 4: Middle cerebral artery Doppler for twin 1.

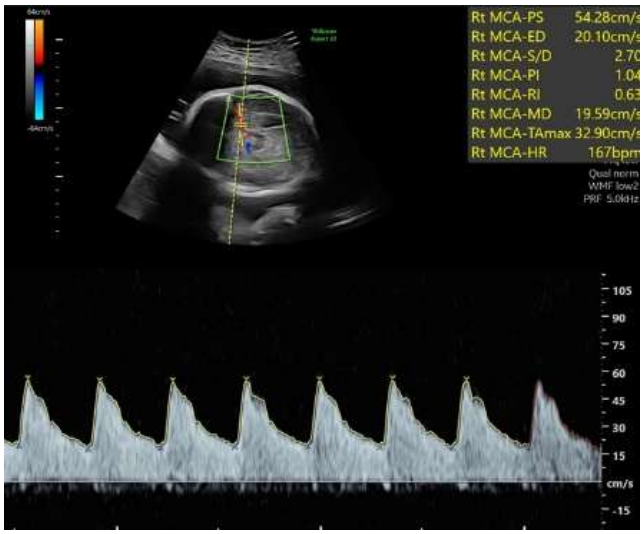

Figure 5: Middle cerebral artery Doppler for twin 1.

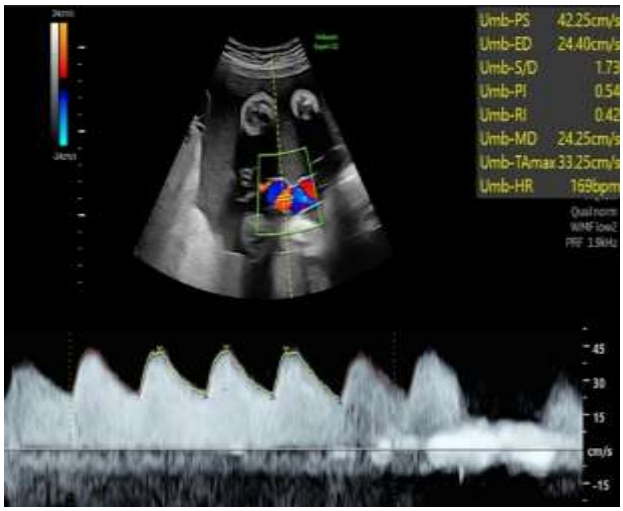

Figure 6: Umbilical artery Doppler for twin 2.

DISCUSSION

Huber et al in his review, has presented the outcomes of twin gestations complicated by TTTS, which are not categorized as severe. His data suggest that amniotic fluid discordance in TTTS leads to adverse perinatal outcomes, if there is associated growth restriction or Doppler abnormalities.[8] In our case, the finding of congenital anemia in the donor twin justified our decision to deliver, nevertheless it has raised a question if earlier intervention should be offered to TTTS.

In a systemic review, the incidence of fetal demise in pregnancies complicated by late TTTS and managed conservatively was triple as compared to gestations where either laser coagulation or amnioreduction was used.[7] In our case, however, there were no neurological sequelae or mortality seen in both twins.

There is a paucity of publications related to maternal renal function in twin gestation. The only information is that twin pregnancies can be related to pre-eclampsia. Attempts have been made to link pre-eclampsia in twin gestation to circulating angiogenic factors.[9]

CONCLUSION

We present a rare case where acute renal injury in the mother developed at the same time as late TTTS. There was no clinical diagnosis of pre-eclampsia. The patient had to be delivered for fetal reasons, but restoration of renal function after birth arguments to some underlying connection. More research is needed to evaluate the impact of TTTS on maternal renal physiology.

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

REFERENCES

1. Society for Maternal-Fetal Medicine, Simpson LL. Twin-twin transfusion syndrome. Am J Obstet Gynecol. 2013;208(1):3-18.
2. Fisher KE, Welsh AW, Wilson I, Henry A. Uncommon complications of monochorionic twin pregnancies: Twin anaemia-polycythaemia sequence. AJUM. 2016;19(2):56-63.
3. Dekonink P, Deprest J, Lewi P, Richer J, Galjaard S, Van Keirsblick J, et al. Gestational age-specific reference ranges for amniotic fluid assessment in monochorionic diamniotic twin pregnancies. Ultrasound Obstet Gynecol. 2013;41(6):649-52.
4. Bronacha MF, Caroline JS, Nirupama H, Sophie L, Shireen M, Christoph CL. Early-onset twin–twin transfusion syndrome: Case series and systematic review. AJUM. 2019;22(4):286-94.
5. Mascio DD, Khalil A, D'Amico A, Buca D, Benedetti PP, Flacco ME, et al. Outcome of twin–twin transfusion syndrome according to Quintero stage of disease: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2020;56(6):811-20.
6. Krispin E, Javinani A, Odibo A, Carreras E, Emery SP, Sepulveda G, et al. Consensus protocol for management of early and late twin–twin transfusion syndrome: Delphi study. Ultrasound Obstet Gynecol. 2024;63(3):371-7.
7. Filomena GS, Francesco D, Benlioglu C, Bhide A, Khalil A. Perinatal outcomes of twin pregnancies complicated by late twin-twin transfusion syndrome: A systematic review and meta-analysis. Acta Obstet Gynecol Scand. 2021;100(5):832-42.
8. Huber A, Diehl W, Zikulnig L, Bregenzer T, Hackeloer BJ, Hecher K. Perinatal outcome in monochorionic twin pregnancies complicated by amniotic fluid discordance without severe twin-twin transfusion syndrome. Ultrasound Obstet Gynecol. 2006;27(1):48-52.
9. Rana S, Michele R, Hacker, Modest AM, Salahuddin S, Lim KH, Verlohren S, et al. Circulating angiogenic factors and risk of adverse maternal and perinatal outcomes in twin pregnancies with suspected preeclampsia. Hypertension. 2012;60(2):451-58.

Cite this article as: Hussain TZ, Ahmed M. A case of late-onset twin-twin transfusion syndrome with severe fetal anemia and maternal acute kidney injury. Int J Reprod Contracept Obstet Gynecol 2025;14:3538-41.