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Case Report

Delayed recognition of twin-to-twin transfusion syndrome with adverse fetal outcome: a case report

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ABSTRACT

Twin-to-Twin Transfusion Syndrome (TTTS) is a serious complication of monochorionic twin pregnancies resulting from imbalanced blood flow through placental vascular anastomoses. It produces a hypovolemic, growth-restricted donor twin and a volume-overloaded recipient twin at risk of cardiac compromise, and carries high perinatal morbidity and mortality if not diagnosed and managed promptly. A 32-year-old unbooked, unsupervised G3P2L2 woman presented at 26 weeks of gestation – at her first antenatal visit – with progressive abdominal distension, breathlessness, reduced urine output, and abdominal pain. Ultrasonography revealed a twin gestation with polyhydramnios in one twin and oligohydramnios with a stuck-twin appearance in the other, along with discordant growth and abnormal Doppler indices, findings compatible with Quintero Stage III TTTS. After counselling regarding all management options, therapeutic amnioreduction was performed for symptomatic relief. Spontaneous preterm labour supervened 14 hours later, and the patient delivered vaginally; the donor twin could not be resuscitated and the recipient twin suffered an early neonatal death. Gross placental examination confirmed monochorionic placentation. This case highlights how absence of early and regular ultrasonographic surveillance in a monochorionic twin pregnancy can delay recognition of TTTS until an advanced, poorly salvageable stage. Routine monochorionic-specific surveillance and prompt referral to a fetal medicine unit capable of fetoscopic laser therapy remain central to improving outcomes.

Keywords: Twin-to-twin transfusion syndrome, Monochorionic diamniotic twins, Fetoscopic laser photocoagulation, Amnioreduction, Quintero staging, Polyhydramnios, Oligohydramnios

INTRODUCTION

Twin-to-Twin Transfusion Syndrome (TTTS) is a serious and potentially fatal condition seen exclusively in monochorionic twin pregnancies, arising from abnormal vascular connections within a shared placenta that produce unequal blood flow between the two fetuses.[1,2] TTTS complicates approximately 10–15% of monochorionic twin pregnancies and typically manifests between 16 and 26 weeks of gestation.[1,2] Risk factors include monochorionic placentation, unequal placental sharing, and absence of compensatory superficial arterio-arterial anastomoses. The underlying mechanism involves persistent transfusion of blood from the donor twin to the recipient twin through deep placental arteriovenous anastomoses.[3,4] Consequently, the donor twin develops reduced circulating blood volume, anaemia, decreased urine production, oligohydramnios, and fetal growth restriction, while the recipient twin experiences volume overload leading to polyuria, polyhydramnios, cardiac strain and, in severe cases, hydrops fetalis. Untreated, advanced-stage TTTS is associated with extremely poor

fetal survival, with reported mortality as high as 80–100%.[5,6] Improvements in prenatal imaging, Doppler velocimetry, and fetal therapeutic interventions have, however, substantially improved perinatal outcomes; among currently available treatments, fetoscopic laser photocoagulation of placental vascular anastomoses is considered the most effective intervention for severe disease.[7,8]

This article describes a case of TTTS that presented late in pregnancy and managed at a tertiary care institution, and discusses its clinical manifestations, diagnostic evaluation, therapeutic challenges, perinatal outcome, and currently recommended evidence-based management strategies.

CASE REPORT

A 32-year-old unbooked, unsupervised woman, G3P2L2, presented to the outpatient department of Obstetrics and Gynaecology, Maharishi Markandeshwar Institute of Medical Sciences and Research, Ambala, at 26 weeks of gestation with progressive abdominal distension, abdominal pain, breathlessness, and decreased urine output. This was her first antenatal visit. She belonged to a lower-middle socio-economic class.

On examination, she appeared uncomfortable because of marked abdominal distension. Pulse rate was 94/min, blood pressure 112/68 mmHg, respiratory rate 20/min, and SpO_2 98% on room air. Fundal height corresponded to approximately 34–36 weeks of gestation. Fetal parts were not palpable because of the excessive liquor volume, and fetal heart sounds could not be localised on auscultation.

Table 1: Baseline Investigations

CBC	**Hb 10 g/dl, TLC 14.3 × 10^3/mm³, Platelets 253 × 10^3/mm³**
LFT	Total bilirubin 1.6 mg/dl, AST 221 U/l, ALT 162 U/l
RFT	Urea 19.2 mg/dl, Creatinine 0.77 mg/dl
Viral markers	Non-reactive
Others	TSH 1.10 µIU/ml; DIPSI 117 mg/dl

Obstetric ultrasonography revealed a live twin pregnancy with a single, anteriorly located placenta. One fetus (Twin A) floated freely within the uterine sac, with normal movements and growth appropriate for gestational age. Marked polyhydramnios was noted, with an amniotic fluid index greater than 30 cm and an estimated fetal weight of approximately 810 g, corresponding to 26 weeks and 1 day of gestation, with an overdistended urinary bladder. The second fetus (Twin B) was stuck to the anterior uterine wall in a prone position, with no visible movement over a prolonged 45-minute observation period, and severe oligohydramnios consistent with the characteristic "stuck-twin" appearance. Its estimated fetal weight was approximately 581 g, corresponding to 23 weeks of gestation, with non-visualisation of the urinary bladder. Colour Doppler showed normal ductus venosus flow and low-resistance umbilical artery flow in Twin A with reversal of a-wave in the ductus venosus, while Twin B showed high-resistance umbilical artery flow with absent end-diastolic velocity and a brain-sparing pattern in the middle cerebral artery.

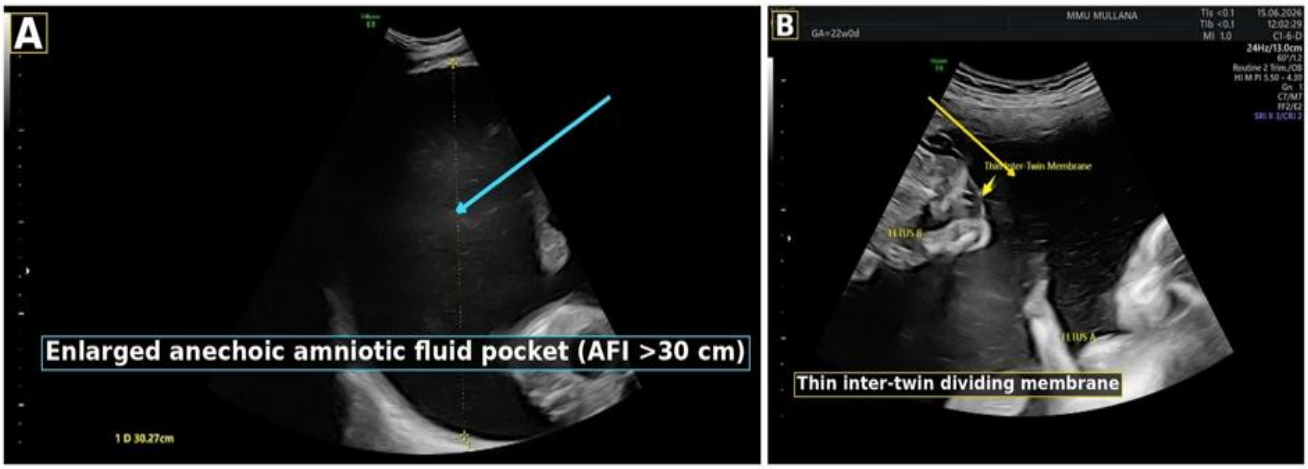

Figure 1: Obstetric ultrasonography at 26 weeks of gestation demonstrating the polyhydramnios-oligohydramnios sequence, (A) Marked polyhydramnios in Twin A (recipient), with an amniotic fluid pocket measuring >30 cm on caliper assessment, (B) Thin inter-twin dividing membrane (arrow) closely applied to Twin B (donor), producing the characteristic "stuck-twin" appearance of severe oligohydramnios

This marked discordance in fetal growth and amniotic fluid volume was highly suggestive of TTTS, and the findings were compatible with Quintero Stage III disease because of the oligohydramnios-polyhydramnios sequence together with abnormal Doppler indices in Twin B. The patient was counselled regarding all management

options, including fetoscopic laser ablation, and was informed of the options available at this institute; however, she opted to continue with a more limited intervention, following which an informed and shared decision for amnioreduction was taken. Therapeutic amnioreduction was performed and approximately 500 mL of amniotic fluid was drained, resulting in transient symptomatic improvement. Despite tocolytics, other precautionary measures, and magnesium sulphate infusion for neuroprotection, the patient developed spontaneous preterm labour 14 hours later and delivered vaginally. Gross examination of the placenta after delivery confirmed monochorionic placentation, consistent with the diagnosis of TTTS.

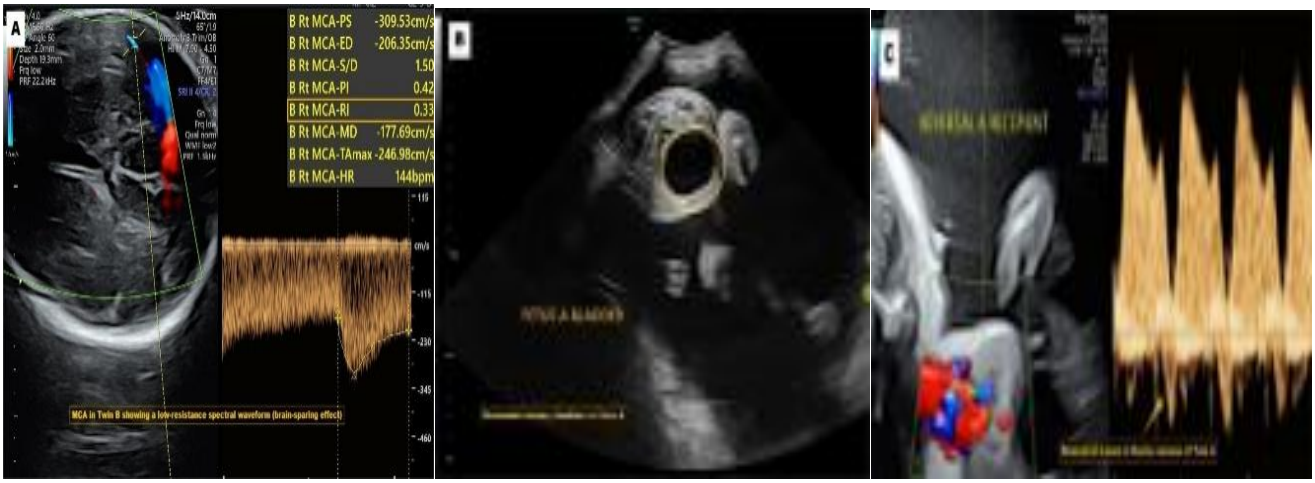

Figure 2: Doppler and structural findings supporting Stage III Twin-to-Twin Transfusion Syndrome, (A) Colour Doppler of the middle cerebral artery in Twin B showing a low-resistance spectral waveform (brain-sparing effect), consistent with fetal hypoxia, (2) Distended urinary bladder in Twin A (recipient), reflecting polyuria from volume overload, (C) Pulsed-wave Doppler of the ductus venosus in Twin A showing intermittent reversal of the a-wave (arrow), a critically abnormal venous Doppler finding.

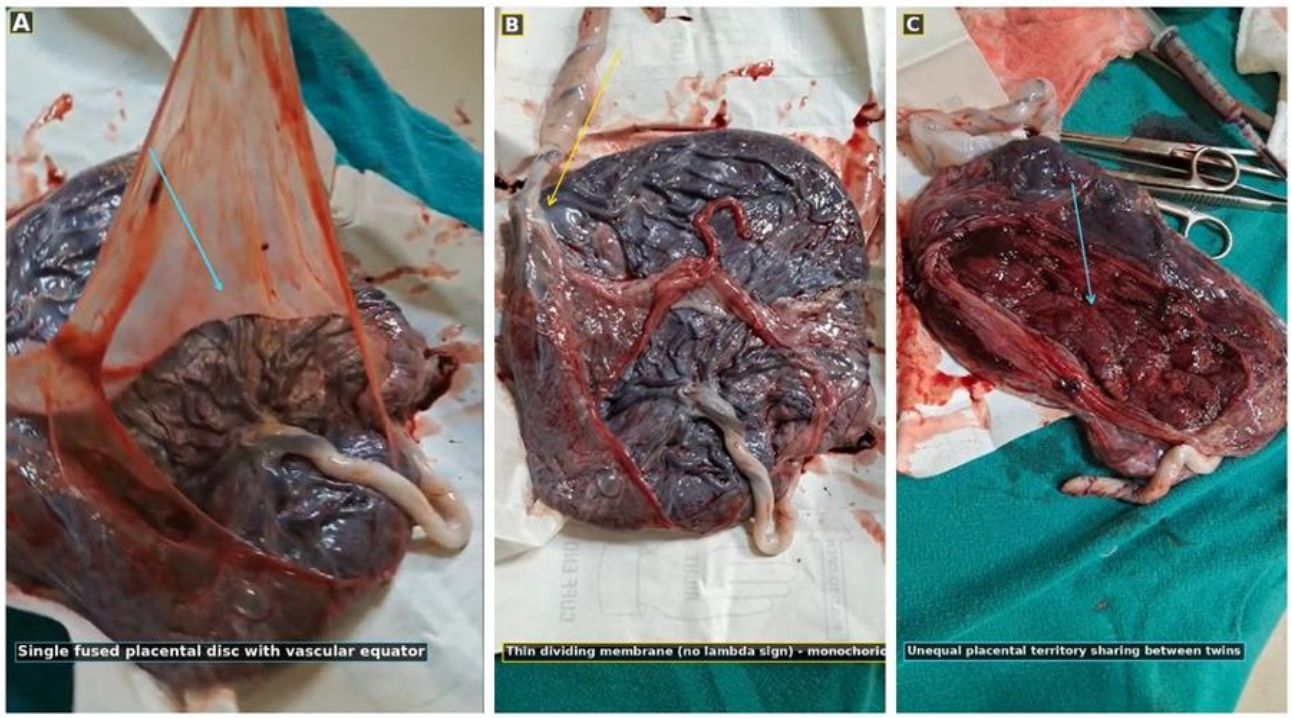

Figure 3: Gross examination of the placenta after delivery, confirming monochorionic placentation, (A) Fetal surface showing a single, fused placental disc with a vascular equator crossing both twins' territories, (B) Thin, translucent dividing membrane without a lambda (twin-peak) sign, characteristic of monochorionicity (C) Grossly unequal distribution of placental territory between the donor and recipient twins.

Twin A, a live male baby delivered in cephalic presentation with excessive liquor, weighed 720 g at birth and was severely depressed, with Apgar scores of 1 and 2 at 1 and 5 minutes respectively. He required immediate resuscitation, intubation, surfactant therapy, and mechanical ventilation. Despite intensive neonatal care, he developed respiratory distress syndrome, neonatal shock requiring multiple inotropes, suspected sepsis, acute kidney injury, and apnoea of prematurity; progressive cardiorespiratory deterioration culminated in refractory shock and cardiopulmonary arrest, resulting in neonatal death.

Twin B, with a birth weight of 596 g, was born with profound birth asphyxia and Apgar scores of 1, 0, and 0 at 1, 5, and 10 minutes respectively. Despite prolonged resuscitation following Neonatal Resuscitation Program (NRP) guidelines, spontaneous circulation could not be achieved, and the neonate was declared dead shortly after birth. The pregnancy thus resulted in neonatal mortality of both twins, attributable primarily to Stage III TTTS complicated by extreme prematurity, severe perinatal asphyxia, and cardiorespiratory failure. This case underscores several key clinical considerations in the management of TTTS. Absence of regular antenatal ultrasonographic monitoring likely contributed to delayed recognition of the condition and its progression to an advanced stage by the time of presentation.

Marked polyhydramnios caused considerable maternal distress and triggered spontaneous preterm labour. The case further highlights the critical role of timely referral to dedicated fetal medicine units, where access to specialised fetal interventions can enhance fetal survival, and underscores that successful management of TTTS requires coordinated multidisciplinary care involving obstetricians, fetal medicine specialists, neonatologists, anaesthesiologists, and neonatal intensive care teams.

DISCUSSION

TTTS is a distinctive complication seen exclusively in monochorionic twin pregnancies, arising from abnormal placental vascular architecture. Unidirectional deep arteriovenous anastomoses facilitate unequal inter-twin blood flow, leading to progressive haemodynamic and haematological imbalance between the fetuses. The donor twin experiences persistent hypovolaemia, resulting in decreased renal perfusion and oliguria, which in turn leads to oligohydramnios. When severe, oligohydramnios or anhydramnios in the donor's sac produces the "stuck-twin" appearance, in which the fetus appears fixed against the uterine wall because there is little or no fluid in its sac and the amniotic membrane lies directly against its body.

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Prenatal ultrasonography is the cornerstone of diagnosis and surveillance in monochorionic pregnancies.[9,10] The diagnostic criteria include a monochorionic diamniotic twin pregnancy with polyhydramnios (maximum vertical pocket >8 cm) in one sac and oligohydramnios (maximum vertical pocket <2 cm) in the other; gestational-age-specific thresholds are also used by some authors (maximum vertical pocket ≥6 cm at 15–17 weeks, ≥8 cm at 18–20 weeks, and >10 cm at ≥20 weeks). Additional criteria include discordant fetal growth, visualisation of the urinary bladder, abnormal Doppler findings, and signs of fetal cardiac dysfunction.

Table 2: Quintero Staging System for TTTS.

Stages	Ultrasound Findings
Stage I	• Oligohydramnios-polyhydramnios sequence. • Bladder of donor twin is visible. • Doppler indices (UA, UV, DV) in both twins are normal.
Stage II	• Oligohydramnios-polyhydramnios sequence. • Bladder of donor twin is not visualised. • Doppler indices (UA, UV, DV) in both twins are normal.
Stage III	• Oligohydramnios-polyhydramnios sequence. • Abnormal Doppler indices: at least one of the following is present in either twin – absent/reversed end-diastolic velocity in the umbilical artery (UA), reversed flow in the a-wave of the ductus venosus (DV), or pulsatile flow in the umbilical vein (UV).
Stage IV	• Oligohydramnios-polyhydramnios sequence. • One or both fetuses show signs of hydrops.
Stage V	• Oligohydramnios-polyhydramnios sequence. • One or both fetuses are dead.

The Quintero staging system remains widely used for prognostication and management planning in TTTS.[4,9] Management of TTTS depends on gestational age,

disease severity, maternal condition, and availability of fetal therapy services.

Expectant management

Mild cases with stable findings may be managed conservatively with close fetal surveillance and regular monitoring; however, the condition can deteriorate rapidly, necessitating timely intervention.

Serial amnioreduction

Amnioreduction reduces intrauterine pressure and provides symptomatic maternal relief by draining excess amniotic fluid from the recipient twin's sac. Although it may reduce the risk of preterm labour associated with severe polyhydramnios, it does not correct the underlying placental vascular pathology.

Fetoscopic laser photocoagulation

Fetoscopic laser ablation of placental vascular anastomoses is currently considered the gold-standard treatment for severe TTTS diagnosed before 26 weeks of gestation. Laser therapy interrupts the abnormal vascular communications and significantly improves survival compared with serial amnioreduction.[1,2,5]

Other Modalities

Septostomy involves creating small perforations in the inter-twin membrane to equalise amniotic fluid volumes; it is used less often because of risks of cord entanglement and membrane rupture. Selective fetal reduction may be considered in severe cases with impending demise of one twin or advanced fetal compromise. Prognosis depends on the stage at diagnosis, gestational age, availability of fetal therapy, and promptness of intervention; untreated severe TTTS carries extremely high fetal mortality. Current evidence demonstrates that fetoscopic laser therapy improves dual survival and neurological outcomes compared with serial amnioreduction.[1-3]

CONCLUSION

Twin-to-Twin Transfusion Syndrome continues to be a major complication of monochorionic twin pregnancies and is associated with considerable fetal morbidity and mortality. Regular antenatal ultrasonographic surveillance plays a crucial role in facilitating early detection and timely management.The present case demonstrates the adverse impact of delayed presentation and restricted availability of advanced fetal therapeutic modalities, and emphasises the need for greater awareness among healthcare professionals and expectant mothers regarding vigilant monitoring of monochorionic pregnancies.Timely referral to specialised tertiary fetal medicine centres, together with coordinated multidisciplinary care and appropriate interventions such as fetoscopic laser photocoagulation, can substantially improve perinatal survival and overall outcomes.

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REFERENCES

1. Society for Maternal-Fetal Medicine; Simpson LL. Twin-twin transfusion syndrome. Am J Obstet Gynecol. 2013;208(1):3-18.
2. Society for Maternal-Fetal Medicine (SMFM); Miller RS, Miller JL, Monson MA, Porter TF, Običan SG, Simpson LL; SMFM Publications Committee. Electronic address: pubs@smfm.org. Society for Maternal-Fetal Medicine Consult Series #72: Twin-twin transfusion syndrome and twin anemia-polycythemia sequence. Am J Obstet Gynecol. 2024;231(4): B16-B37.
3. Ville Y, Hecher K, Gagnon A, Sebire N, Hyett J, Nicolaides K. Endoscopic laser coagulation in the management of severe twin-to-twin transfusion syndrome. Br J Obstet Gynaecol. 1998;105(4):446-53.
4. Quintero RA, Morales WJ, Allen MH, Bornick PW, Johnson PK, Kruger M. Staging of twin-twin transfusion syndrome. J Perinatol. 1999;19(8 Pt 1):550-5.
5. Senat MV, Deprest J, Boulvain M, Paupe A, Winer N, Ville Y. Endoscopic laser surgery versus serial amnioreduction for severe twin-to-twin transfusion syndrome. N Engl J Med. 2004;351(2):136-44.
6. Roberts D, Gates S, Kilby M, Neilson JP. Interventions for twin-twin transfusion syndrome: a Cochrane review. Ultrasound Obstet Gynecol. 2008;31(6):701-11.
7. Zwet EW, Weingertner AS, Klumper FJ, DeKoninck P, Devlieger R, Kilby MD et al. Fetoscopic laser coagulation of the vascular equator versus selective coagulation for twin-to-twin transfusion syndrome: an open-label randomised controlled trial. Lancet. 2014;383(9935):2144-51.
8. Robyr R, Lewi L, Salomon LJ, Yamamoto M, Bernard JP, Deprest J et al. Prevalence and management of late fetal complications following successful selective laser coagulation of chorionic plate anastomoses in twin-to-twin transfusion syndrome. Am J Obstet Gynecol. 2006;194(3):796-803.
9. Khalil A, Rodgers M, Baschat A, Bhide A, Gratacos E, Hecher K et al. ISUOG Practice Guidelines: role of ultrasound in twin pregnancy. Ultrasound Obstet Gynecol. 2016;47(2):247-63.
10. Lewi L, Gucciardo L, Huber A, Jani J, Van Mieghem T, Doné E, Cannie M, Gratacós E, Diemert A, Hecher K, Lewi P, Deprest J. Clinical outcome and placental characteristics of monochorionic diamniotic twin pairs with early- and late-onset discordant growth. Am J Obstet Gynecol. 2008;199(5):511.e1-7.

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