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Spontaneous Twin Anemia–Polycythemia Sequence (TAPS): a complication of monochorionic twin pregnancy

Abstract

Twin anemia–polycythemia sequence (TAPS) is a rare but serious complication unique to monochorionic twin pregnancies. It results from chronic, slow transfusion of blood through a limited number of very small placental vascular anastomoses, leading to progressive anemia in the donor twin and polycythemia in the recipient twin. Unlike twin-to-twin transfusion syndrome (TTTS), TAPS typically occurs in the absence of amniotic fluid discordance, making diagnosis more challenging.

The underlying pathophysiology is characterized by an unbalanced placental vascular architecture consisting predominantly of minute arteriovenous anastomoses with absent or insufficient compensatory arterioarterial connections. Antenatal diagnosis relies primarily on Doppler assessment of the middle cerebral artery peak systolic velocity (MCA-PSV), while postnatal confirmation is based on intertwin hemoglobin differences, reticulocyte count ratios, and placental examination.

Management strategies include expectant monitoring, intrauterine transfusion, partial exchange transfusion, and fetoscopic laser coagulation. Among these options, laser coagulation is the only treatment that addresses the underlying placental vascular pathology. The introduction of the Solomon laser technique has significantly reduced the incidence of residual anastomoses and post-laser TAPS.

Despite advances in diagnostic criteria and therapeutic approaches, TAPS remains underdiagnosed and poses significant challenges in clinical management. Further research is needed to optimize screening protocols, refine classification systems, and establish evidence-based treatment guidelines to improve perinatal outcomes in affected pregnancies.

Keywords: monochorionic twins, twin anemia–polycythemia sequence, TAPS, fetal anemia, fetal therapy, placental vascular anastomoses

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Abbreviations: AA, arterioarterial anastomoses; AV, Arteriovenous anastomoses; CDR, color difference ratio; CRL, crown-rump length; IUT, intrauterine transfusion; MC, Monochorionic; MCA-PSV, middle cerebral artery peak systolic velocity; MeSH, medical subject headings; PET, partial exchange transfusion; TAPS, twin anemia–polycythemia sequence; TRAP, twin reversed arterial perfusion sequence; TTS, twin-to-twin transfusion syndrome; US, ultrasound; VV, venovenous anastomoses

Introduction

Over the past three decades, the incidence of multiple pregnancies has risen markedly, largely due to increased use of assisted reproductive technologies. Multiple gestations pose substantial clinical challenges. They are associated with significantly higher perinatal morbidity and mortality, including an increased risk of long-term neurodevelopmental impairment, compared to single pregnancies. They also exhibit higher rates of preterm birth and intrauterine growth restriction, both of which contribute considerably to adverse perinatal outcomes.[1–3]

The true incidence of multiple pregnancies is difficult to estimate, as many end in early miscarriage. Additionally, there is the possibility of a vanishing twin in the early embryonic phase, where one embryo may vanish due to various factors. As a result, the pregnancy may continue with one or two fetuses, depending on the initial number of embryos. Literature estimates that approximately 1 in 89 conceptions results in a twin pregnancy. Additionally, the use of assisted reproductive techniques has drastically increased the global incidence of multiple pregnancies.[2,3–5]

Monochorionic (MC) twin pregnancies are associated with increased perinatal morbidity and mortality due to shared placental vascular anastomoses. Characteristic complications include twin-to-twin transfusion syndrome (TTTS), twin anemia–polycythemia sequence (TAPS), twin reversed arterial perfusion (TRAP) sequence, monoamniotic pregnancy, and conjoined twinning.[2,3–5]

Spontaneous twin anemia polycythemia sequence (TAPS) is a complication of MC twin pregnancies characterized by highly discordant hemoglobin values between the twins (polycythemic recipient and anaemic donor). In contrast to TTTS, both twins have normal amniotic fluid volume in the classic, pure form of TAPS.[6] TAPS complicates about 3-5% MC diamniotic twin pregnancies and is a complication of the third trimester which usually occurs after 26 weeks of gestation (weeks of gestation). Iatrogenic TAPS can occur after laser treatment for TTTS. Post-laser TAPS occurs in 2–16% of TTTS cases after incomplete laser. The wide range in incidence rate in post-laser TAPS can be explained by the use of different laser surgical techniques and/or the existence of different definitions and criteria for TAPS.[4,6]

Although significant advances have been made in understanding the pathophysiology, diagnosis, and management of TAPS, important

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challenges remain regarding optimal screening, classification, and treatment strategies. This review summarizes current evidence regarding placental characteristics, pathophysiological mechanisms, diagnostic approaches, classification systems, and contemporary management options for TAPS.

Methods

A narrative literature review was conducted to summarize current evidence regarding the pathophysiology, diagnosis, classification, management, and outcomes of twin anemia–polycythemia sequence (TAPS) in monochorionic twin pregnancies.

A comprehensive search of the PubMed/MEDLINE, Scopus, and Google Scholar databases was performed for articles published between January 2000 and January 2026. The following keywords and Medical Subject Headings (MeSH) terms were used alone and in combination: "twin anemia polycythemia sequence," "TAPS," "monochorionic twins," "monochorionic pregnancy," "placental vascular anastomoses," "twin-to-twin transfusion syndrome," "middle cerebral artery peak systolic velocity," "fetoscopic laser coagulation," "intrauterine transfusion," and "Solomon technique."

Reference lists of relevant articles, review papers, clinical guidelines, and consensus statements were additionally screened to identify further eligible publications.

Study selection

Priority was given to original research articles, systematic reviews, meta-analyses, consensus statements, international practice guidelines, and major cohort studies addressing TAPS pathophysiology, diagnosis, classification, treatment, and perinatal outcomes. Publications written in English were included.

Case reports and small case series were reviewed when they provided important information regarding rare clinical presentations, diagnostic findings, or therapeutic approaches. Duplicate publications and studies lacking sufficient methodological detail were excluded where appropriate.

The findings were synthesized narratively to provide an updated overview of current knowledge and ongoing controversies regarding TAPS management and diagnosis.

Characteristics of the MC placenta in TAPS

In monochorionic (MC) placentas, vascular communications arise where the fetal circulations of both twins meet at the boundary between their respective placental territories. These vascular connections may occur superficially on the placental surface or through deeper shared placental cotyledons. The vascular equator represents the boundary between the two placental territories and contains the vascular anastomoses responsible for intertwin blood exchange. The shared placental circulation, often referred to as the "third circulation," accounts for approximately 5–10% of the vascular volume of each twin.[2,3–5]

Placental vascular anastomoses vary in type, number, and size. Arteriovenous (AV) anastomoses connect an artery from one twin to a vein of the co-twin through a shared cotyledon, resulting in unidirectional blood transfer. In contrast, arterioarterial (AA) and venovenous (VV) anastomoses are superficial connections that permit bidirectional flow according to pressure gradients. In most MC pregnancies, these vascular exchanges remain balanced.[2,3–5]

TAPS placentas are characterized by fewer vascular connections than uncomplicated MC placentas or those affected by TTTS. The vascular architecture is dominated by small AV anastomoses, while AA and VV connections are uncommon. AA anastomoses are present in only 14% of TAPS cases and are markedly smaller than those observed in uncomplicated MC placentas. Since large AA anastomoses are common in normal MC pregnancies, their presence may protect against the development of TAPS by compensating for unbalanced intertwin transfusion.[6–8]

TAPS is exceptionally rare in monoamniotic twins This rarity is likely related to the presence of large vascular communications and closely positioned umbilical cord insertions that facilitate hemodynamic equilibration between the twins. Nevertheless, isolated cases have been reported in monoamniotic twins in which only minute AV anastomoses were identified during fetoscopic evaluation.[9]

Placental sharing does not appear to differ substantially between spontaneous TAPS and uncomplicated MC pregnancies. Interestingly, although donor twins are usually smaller at birth, they often possess a larger placental share, suggesting that fetal growth in TAPS is more strongly influenced by donor–recipient status than by placental territory.[6,10]

The relationship between TAPS and velamentous cord insertion remains uncertain. Although some studies have reported an increased prevalence of velamentous insertion in TAPS pregnancies, larger investigations have not consistently confirmed this association.[6,10]

A characteristic pathological finding of TAPS placentas is the marked difference in placental appearance between twins. The placental territory of the anemic donor twin typically appears pale, whereas the polycythemic recipient's share is dark and congested. Similar differences may be observed prenatally on ultrasound, with increased echogenicity in the donor placental territory and relatively reduced echogenicity in the recipient's placental share.[6,11]

Pathophysiology of TAPS

Twin anemia–polycythemia sequence is caused by chronic, slow transfusion of blood from the donor twin to the recipient twin through a limited number of extremely small placental vascular anastomoses. Unlike TTTS, which is characterized by significant intertwin fluid imbalance, TAPS primarily results from a progressive transfer of red blood cells, producing severe hematological discordance while amniotic fluid volumes remain normal.[4,12]

The vascular architecture of TAPS is unique. Most placentas contain only a few AV anastomoses, typically measuring less than 1 mm in diameter. These minute vascular connections permit a slow but persistent transfusion of approximately 5–15 mL of blood per day from the donor to the recipient twin. Over time, this chronic transfusion results in progressive anemia in the donor and polycythemia in the recipient.[8,12–14]

Compared with uncomplicated monochorionic placentas, TAPS placentas contain significantly fewer vascular anastomoses overall. While normal monochorionic placentas may contain approximately eight vascular connections, TAPS placentas typically contain only three to four.[7,12–14]

AA anastomoses are present in only 10–20% of TAPS cases and are usually considerably smaller than those observed in uncomplicated monochorionic pregnancies or TTTS. Because AA anastomoses

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allow bidirectional blood flow, they are believed to protect against unbalanced transfusion and reduce the risk of both TTTS and TAPS.[13,14]

Differences have also been observed between spontaneous and post-laser TAPS. Spontaneous TAPS placentas generally contain a greater number of vascular anastomoses than post-laser TAPS placentas. Post-laser TAPS develops when residual placental vascular connections remain following incomplete fetoscopic laser coagulation for TTTS. Although the number of residual anastomoses is small, they may still permit chronic intertwin transfusion sufficient to produce severe hematological imbalance.[7,12–14]

Diagnosis of TAPS

Accurate determination of chorionicity is fundamental in the assessment of twin pregnancies because TAPS occurs exclusively in monochorionic gestations. Ultrasound (US) examination performed between 11+0 and 13+6 weeks of gestation provides the highest diagnostic accuracy for determining chorionicity and amnionicity. The ideal period is between 11+0-13+6 weeks of gestation. US details that are important to note in the first trimester are: determining crown-rump length- CRL, location of placenta/placentas and characteristics of the amniotic membranes. If the pregnancy is >14 weeks of gestation, gestational age is determined by measuring head circumference. Determining chorionicity and amnionicity in twin pregnancy is a necessity. With US <13+6 weeks of gestation chorionicity is identified with approximately 95% accuracy. The presence of two placentas is the detection of the characteristic "lambda" or "delta" sign (thickening at the site of membrane insertion). An additional US sign is the presence of 2 placental masses. Detection of different sexes of the fetuses indicates dizygotic pregnancy. Amnionicity is determined by the so-called T-sign (presence of an intraamniotic septum).[2,3,15–17]

TAPS may be diagnosed antenatally or postnatally. It is characterized by significant intertwin hemoglobin discordance in the absence of the classic amniotic fluid abnormalities seen in TTTS. Antenatal diagnosis relies primarily on Doppler ultrasound findings, whereas postnatal diagnosis is based on hematologic imbalance between twins and confirmatory placental findings.[4,13]

Antenatal diagnosis

The middle cerebral artery peak systolic velocity (MCA-PSV) is the main non-invasive tool for detecting fetal anemia and polycythemia. In TAPS, the donor twin typically shows elevated MCA-PSV consistent with anemia, while the recipient demonstrates reduced velocities suggestive of polycythemia.[4,18]

Diagnostic thresholds have evolved over time. Initial criteria proposed MCA-PSV >1.5 MoM in the donor and <0.8 MoM in the recipient. Subsequent data indicated that values between 0.8 and 1.0 MoM may also be associated with postnatally confirmed cases, leading to the currently used cut-offs of >1.5 MoM for the donor and <1.0 MoM for the recipient. These thresholds show good sensitivity and specificity in selected high-risk cohorts from specialized fetal therapy centers.[4,13,18]

However, subsequent studies have questioned the reliability of MCA-PSV for detecting polycythemia, reporting no significant difference in absolute values between polycythemic and uncomplicated fetuses. A stronger correlation was found between intertwin differences in MCA-PSV (delta (Δ) MCA-PSV) and hematocrit discrepancy. Based on these findings, a ΔMCA-PSV >0.5 MoM has been proposed as an alternative diagnostic approach.[4,13,18]

Practical application and borderline cases: In routine surveillance, clinicians should consider both absolute MCA-PSV and delta MCA-PSV values. When delta MCA-PSV is >0.5 MoM but absolute values are within normal limits, close monitoring is warranted, as this may indicate early or mild TAPS. Conversely, if delta MCA-PSV is borderline (e.g., 0.4–0.5 MoM), repeat Doppler assessment within 24–48 hours is recommended to confirm or exclude progression. In cases of persistent borderline results, additional parameters such as fetal growth discordance, amniotic fluid volume, or fetal biometry may provide further clarity.[13,18]

TAPS staging reflects disease progression, although this progression may be non-linear and can occasionally regress. The table below presents the prenatal staging criteria according to both the traditional and delta MCA-PSV systems. A staging system is essential for distinguishing the varying degrees of TAPS severity, facilitating standardized comparison of cases, and guiding treatment decisions across centers.[13,18]

Additional antenatal ultrasound features have been described in some cases, including discordant placental thickness and echogenicity, reflecting the pale, edematous appearance of the anemic placental share versus the relatively normal appearance of the polycythemic share. A "starry sky" pattern of the fetal liver has also been reported, although this finding is nonspecific and may occur in other conditions such as hepatic inflammation or cardiac failure. Further studies are required to clarify the diagnostic value of these additional sonographic signs.[13,19]

Given the association between early diagnosis and improved outcomes, regular (at least biweekly) MCA-PSV surveillance is recommended in monochorionic pregnancies for timely detection of TAPS.[13,20]

In the study of Khalil A.et al, consensus-based diagnostic criteria for TAPS were established using a Delphi process. Antenatally, TAPS was defined by MCA-PSV values ≥1.5 MoM in the anemic twin and ≤1.0 MoM (previously ≤0.8 MoM) in the polycythemic twin, or alternatively by an intertwin MCA-PSV difference ≥1.0 MoM.[21]

The panel further agreed that surveillance for TAPS should be performed at two-week intervals, and that disease severity should be graded using an antenatal classification system based on MCA-PSV measurements. Consensus was also reached on key parameters relevant for both antenatal monitoring and postnatal follow-up in affected pregnancies.[21]

Postnatal diagnosis

A substantial proportion of TAPS cases (approximately 40–60%) are not detected antenatally and are diagnosed only after birth. Postnatal diagnosis is based on intertwin hemoglobin discordance and placental examination.[7,13]

Earlier definitions used absolute hemoglobin thresholds. However, these do not account for gestational age-related increases in fetal hemoglobin. Gestational age-adjusted criteria (e.g., hemoglobin <5th percentile in the donor and hematocrit >65% in the recipient) improved accuracy but were limited by the need for reference charts. Consequently, a fixed intertwin hemoglobin difference >8 g/dL is now commonly used as a pragmatic diagnostic criteria.[13,22–25]

A major diagnostic challenge is differentiation between TAPS and acute peripartum TTTS, as both conditions may present with marked hemoglobin discordance at birth. Two additional parameters assist in distinguishing them. First, an elevated reticulocyte count ratio (>1.7

between donor and recipient) reflects chronic erythropoietic response and is considered characteristic of TAPS. In contrast, acute TTTS typically shows low or normal reticulocyte levels due to the short time interval between transfusion and delivery.[13,25]

Second, placental dye injection may demonstrate the presence of small residual anastomoses (<1 mm), supporting a diagnosis of TAPS. Acute TTTS, in contrast, is usually associated with large, low-resistance arterioarterial or venovenous anastomoses.[13,22,25]

Because reticulocyte measurements and placental injection studies are not always available in clinical practice, additional diagnostic approaches have been explored. One proposed method is assessment of a placental color difference ratio (CDR) based on digital analysis of the maternal placental surface. TAPS placentas demonstrate a significantly higher CDR (>1.5) compared with uncomplicated monochorionic placentas. However, further validation in larger cohorts, including cases of acute TTTS, is required before this method can be incorporated into routine diagnostic criteria.[13]

Regarding Khalil A.et al.study, postnatal diagnosis was based on an intertwin hemoglobin difference ≥8 g/dL together with a reticulocyte count ratio ≥1.7.[21]

Classification of TAPS

Because TAPS encompasses a wide spectrum of disease severity, standardized classification systems have been developed to facilitate diagnosis, guide management, and allow comparison of outcomes across studies and fetal therapy centers.[22–25]

Traditionally, antenatal staging of twin anemia-polycythemia sequence (TAPS) relied on absolute middle cerebral artery peak systolic velocity (MCA-PSV) values. Donor twins with MCA-PSV values greater than 1.5 multiples of the median (MoM) were classified as anemic, while recipient twins with values below 1.0 MoM were considered polycythemic. However, these criteria, though highly specific, demonstrated relatively low sensitivity for detecting all clinically relevant cases of TAPS, particularly in early or mild disease stages.[26,27]

To address this limitation, Tollenaar et al. (2018) introduced a revised classification based on the intertwin difference in MCA-PSV (delta MCA-PSV), with a diagnostic cutoff of >0.5 MoM. This approach significantly improves sensitivity to 83% while maintaining 100% specificity, enabling the identification of TAPS even when one twin's MCA-PSV remains within the normal range. The delta MCA-PSV method is particularly useful in cases where absolute MCA-PSV values are borderline or equivocal, as it accounts for the physiological differences between twins and reduces the risk of false negatives.[28–30]

Prenatal and Postnatal Staging: Because TAPS presents with varying degrees of severity, a staging system is useful for distinguishing its different forms. Such a classification also facilitates standardized comparison of cases and treatment outcomes across centers. For this reason, both prenatal and postnatal staging systems have recently been proposed. Similar to twin-twin transfusion syndrome (TTTS), TAPS progression is not always linear and may regress. The staging system proposed by Tollenaar et al. (2018) incorporates both prenatal and postnatal criteria, with prenatal stages defined by MCA-PSV and delta MCA-PSV values, as outlined in Table 1.[13,29,30]

Table 1 Antenatal TAPS classification[28–30]

Antenatal stage	Finding at Doppler ultrasound examination
Stage 1	Delta MCA-PSV > 0.5 MoM, without signs of fetal compromise
Stage 2	Delta MCA-PSV > 0.7 MoM, without signs of fetal compromise
Stage 3	As Stage 1 or 2, with cardiac compromise of donor
Stage 4	Hydrops of donor
Stage 5	Intrauterine demise of one or both fetuses preceded by TAPS

This staging system reflects increasing disease severity and helps guide clinical decision-making.

Postnatal classification is based primarily on hematological findings and placental examination. Severity is determined according to the degree of intertwin hemoglobin discordance, reticulocyte count ratio, and evidence of placental vascular anastomoses. Consensus criteria established through an international Delphi process define postnatal TAPS as:[23,29,30]

a) Intertwin hemoglobin difference ≥8 g/dL.

b) Reticulocyte count ratio ≥1.7.

c) Evidence of small placental vascular anastomoses.

The incorporation of both hematologic and placental criteria improves diagnostic accuracy and assists in differentiating TAPS from acute peripartum twin-to-twin transfusion.

Perinatal management and outcomes

The optimal management of twin anemia-polycythemia sequence (TAPS) remains uncertain. Available approaches include expectant management, intrauterine transfusion (IUT), partial exchange transfusion (PET), fetoscopic laser coagulation, selective feticide, and timely delivery when indicated. Management should be individualized according to disease severity, gestational age, and available treatment options.[13,31]

Expectant management involves intensive ultrasound surveillance, including serial assessment of middle cerebral artery peak systolic velocity (MCA-PSV). This approach may be appropriate for mild cases, particularly stages 1 and 2, although close monitoring is essential due to the risk of progression. For conservatively managed TAPS, delivery is typically recommended at 34–36 weeks' gestation to balance the risks of fetal deterioration against prematurity, unless earlier delivery is warranted by worsening fetal status (e.g., abnormal Doppler findings, hydrops, or non-reassuring fetal testing). Advanced disease or rapid deterioration generally warrants intervention or delivery, balancing fetal benefits against the risks of prematurity.[13,31]

Intrauterine Transfusion (IUT) is commonly used to correct anemia in the donor twin and may be administered intravascularly or intraperitoneally, with the latter potentially reducing rapid transfusion-related blood shifts to the recipient. However, IUT does not address the underlying placental vascular connections and therefore provides only temporary improvement. In addition, correction of donor anemia may worsen polycythemia and hyperviscosity in the recipient twin.[13,32–34]

To minimize this risk, IUT can be combined with PET in the recipient twin, reducing blood viscosity and improving hematologic balance. Computational and clinical studies suggest that this combined approach may offer advantages over IUT alone. An alternative strategy involves using blood obtained from the polycythemic co-twin during PET to transfuse the anemic donor, thereby avoiding exposure to donor blood products. Although promising, further studies are needed to evaluate its impact on clinical outcomes.[13,32,34]

Fetoscopic laser coagulation is currently the only treatment that directly targets the underlying cause of TAPS by interrupting placental vascular anastomoses. The procedure is technically more challenging than laser treatment for twin-to-twin transfusion syndrome (TTTS) because TAPS is not associated with oligohydramnios-polyhydramnios sequence, making visualization of the vascular equator more difficult. Furthermore, the placental connections involved are often few and extremely small.[13,34–36]

Available studies indicate that laser therapy can prolong pregnancy and improve perinatal outcomes compared with expectant management or IUT alone. Following successful laser coagulation, delivery is generally deferred until at least 34 weeks' gestation, provided fetal surveillance remains reassuring. If residual or recurrent TAPS is suspected post-laser, delivery timing should be individualized based on disease severity and gestational age. To reduce the risk of residual vascular connections and recurrent disease, the Solomon technique is recommended, in which a continuous laser line is created between individual coagulation sites across the placental equator.[13,34–36]

Although spontaneous resolution of TAPS has been reported, it appears to be rare and should not be expected in routine clinical practice.[13,34,37,38]

Suggested antenatal management: In the absence of definitive evidence, management should be guided by TAPS stage, gestational age, and treatment feasibility. Stages 1 and selected stage 2 cases may be managed conservatively with close surveillance. For conservatively managed cases, planned delivery at 34–36 weeks is advised, with earlier delivery indicated for fetal compromise. Intervention should be considered for rapidly progressive disease or stages 3 and above. Before 28 weeks' gestation, fetoscopic laser surgery is generally preferred when technically feasible because it treats the underlying pathology and may prolong pregnancy. If laser treatment is performed after 28 weeks, delivery is typically planned at 34–36 weeks, unless earlier delivery is required due to persistent or recurrent TAPS. If laser treatment is not possible, IUT may be considered, particularly before 30–32 weeks. PET may be added when severe recipient polycythemia is present or when repeated transfusions are anticipated.[13,35,36]

Prevention of post-laser TAPS: Post-laser TAPS results from residual placental anastomoses following fetoscopic laser treatment for TTTS. The Solomon technique was developed to reduce this complication and has been shown to significantly decrease the incidence of post-laser TAPS without increasing short-term adverse outcomes.

Long-term follow-up studies have demonstrated comparable neurodevelopmental outcomes at 2 years of age between Solomon-treated and conventionally treated children, with the majority of TAPS survivors showing normal cognitive and motor development. However, subtle neurodevelopmental delays have been reported in a subset of cases, particularly those with advanced TAPS stages (≥3), severe anemia or polycythemia, or a history of intrauterine demise of a co-twin. Consequently, longitudinal neurodevelopmental follow-up is recommended for all TAPS survivors, especially those with a complicated perinatal course.[14,15,34,37]

In addition to neurodevelopmental considerations, hematological sequelae are common in TAPS neonates. Polycythemia in the recipient twin may lead to hyperviscosity syndrome, increasing the risk of thromboembolic events or necrotizing enterocolitis if untreated[18]. Severe anemia in the donor twin often requires postnatal blood transfusion and may be associated with neonatal morbidity, such as respiratory distress or feeding difficulties[19]. Iron supplementation is frequently recommended for anemic donors to support hematologic recovery, though optimal duration remains uncertain. The summary of the key outcomes is shown in the Table 2.[20,37,38]

Table 2 Summary of key outcomes

Outcome	Findings
Neurodevelopmental	Mostly normal; subtle delays in advanced TAPS (≥Stage 3)
Hematological	Polycythemia (recipient), anemia (donor), iron supplementation recommended
Follow-up Recommendations	Longitudinal monitoring for all survivors, especially with complications

The Solomon technique is recommended during laser surgery for TTTS to minimize residual vascular connections and reduce the risk of subsequent TAPS, without adversely affecting long-term neurodevelopmental outcomes.[2,13,21,22,37,39]

Conclusion

Twin anemia–polycythemia sequence is a unique complication of monochorionic twin pregnancy resulting from chronic, low-volume transfusion through a limited number of small placental vascular anastomoses. Unlike twin-to-twin transfusion syndrome, TAPS is characterized by significant hematological discordance without the typical amniotic fluid abnormalities, making diagnosis more challenging.

Middle cerebral artery peak systolic velocity assessment has become the cornerstone of antenatal diagnosis, while postnatal confirmation relies on hematological findings and placental examination. Increasing evidence supports the use of intertwin MCA-PSV differences as a more sensitive diagnostic marker.

Management should be individualized according to disease stage, gestational age, and available expertise. Although expectant management and intrauterine transfusion remain important therapeutic options, fetoscopic laser coagulation is currently the only treatment that directly addresses the underlying placental pathology. The introduction of the Solomon technique has substantially reduced the incidence of residual vascular anastomoses and post-laser TAPS.

Despite considerable advances in understanding the disease, important questions remain regarding optimal screening, staging, and treatment. Continued collaborative research and international consensus efforts are essential to improve diagnosis, standardize management, and optimize perinatal and long-term outcomes for affected twins.

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Conflicts of interest

The authors declare no conflicts of interest.

References

1. Oliver E, Navaratnam K, Gent J, et al. Comparison of international guidelines on the management of twin pregnancy. *Eur J Obstet Gynecol Reprod Biol*. 2023;285:97–104.
2. Sileo FG, Sorrenti S, Giancotti A, et al. Counselling in monochorionic twin pregnancies. *J Clin Med*. 2024;13(23):7355.
3. Chalouhi GE, Stirnemann JJ, Salomon LJ, et al. Specific complications of monochorionic twin pregnancies: twin-twin transfusion syndrome and twin reversed arterial perfusion sequence. *Semin Fetal Neonatal Med*. 2010;15(6):349–356.
4. Coady AM, Bower S. *Twinings textbook of fetal abnormalities*. 3rd ed. Elsevier Health Sciences; 2014.
5. Simpson LL, Miller RS. Twin-twin transfusion syndrome. In: Copel JA, D'Alton ME, Feltovich H, et al, editors. *Obstetric Imaging: Fetal Diagnosis and Care*. 2nd ed. Elsevier; 2018.
6. Couck I, Lewi L. The placenta in twin-to-twin transfusion syndrome and twin anemia polycythemia sequence. *Twin Res Hum Genet*. 2016;19(3):184–190.
7. de Villiers SF, Slaghekke F, Middeldorp JM, et al. Placental characteristics in monochorionic twins with spontaneous versus post-laser twin anemia-polycythemia sequence. *Placenta*. 2013;34(5):456–459.
8. de Villiers SF, Zhao DP, Cohen D, et al. Correlation between veno-venous anastomoses, TTTS and perinatal mortality in monochorionic twin pregnancies. *Placenta*. 2015;36(5):603–606.
9. Diehl W, Glosemeyer P, Tavares de Sousa M, et al. Twin anemia–polycythemia sequence in monoamniotic twins. *Ultrasound Obstet Gynecol*. 2013;42(1):108–111.
10. Zhao DP, Cohen D, Middeldorp JM, et al. The role of veno-venous anastomoses in twin-twin transfusion syndrome. *Placenta*. 2014;35(5):334–336.
11. Movva VC, Rijhsinghani A. Discrepancy in placental echogenicity: a sign of twin anemia polycythemia sequence. *Prenat Diagn*. 2014;34(8):809–811.
12. Zhao DP, de Villiers SF, Slaghekke F, et al. Prevalence, size, number and localization of vascular anastomoses in monochorionic placentas. *Placenta*. 2013;34(7):589–593.
13. Tollenaar LS, Slaghekke F, Middeldorp JM, et al. Twin anemia polycythemia sequence: current views on pathogenesis, diagnostic criteria, perinatal management, and outcome. *Twin Res Hum Genet*. 2016;19(3):222–233.
14. Tollenaar LSA, Lopriore E, Oepkes D, et al. Twin anemia polycythemia sequence: knowledge and insights after 15 years of research. *Matern Fetal Med*. 2021;3(1):33–41.
15. Leombroni M, Liberati M, Fanfani F, et al. Diagnostic accuracy of ultrasound in detecting birth-weight discordance in twin pregnancy. *Ultrasound Obstet Gynecol*. 2017;50(4):442–450.
16. ISUOG Practice Guidelines: ultrasound assessment of fetal biometry and growth. *Ultrasound Obstet Gynecol*. 2019;53(6):715–723.
17. ISUOG Practice Guidelines: role of ultrasound in twin pregnancy. *Ultrasound Obstet Gynecol*. 2025.
18. Fishel-Bartal M, Weisz B, Mazaki-Tovi S, et al. Can MCA-PSV predict polycythemia in monochorionic twins? *Ultrasound Obstet Gynecol*. 2016;48(4):470–475.
19. Soundararajan LP, Howe DT. Starry sky liver in twin anemia-polycythemia sequence. *Ultrasound Obstet Gynecol*. 2014;43(5):601–602.
20. Rossi AC, Prefumo F. Perinatal outcomes of twin anemia–polycythemia sequence: a systematic review. *J Obstet Gynaecol Can*. 2014;36(8):701–707.
21. Khalil A, Gordijn S, Ganzevoort W, et al. Consensus diagnostic criteria and monitoring of twin anemia–polycythemia sequence: Delphi procedure. *Ultrasound Obstet Gynecol*. 2020;56(3):388–394.
22. Lewi L, Jani J, Blickstein I, et al. The outcome of monochorionic diamniotic twin gestations in the era of invasive fetal therapy. *Am J Obstet Gynecol*. 2008;199(5):514.e1–514.e8.
23. Jopling J, Henry E, Wiedmeier SE, et al. Reference ranges for hematocrit and hemoglobin in neonates. *Pediatrics*. 2009;123(2):e333–e337.
24. Nicolaides KH, Thilaganathan B, Mibashan RS. Cordocentesis in fetal erythropoiesis. *Am J Obstet Gynecol*. 1989;161(5):1197–1200.
25. Lopriore E, Slaghekke F, Middeldorp JM, et al. Accute and simple evaluation of vascular anastomoses using dye injection. *J Vis Exp*. 2011;(55):3208.
26. Khalil A, Rodgers M, Baschat A, et al. ISUOG Practice Guidelines: role of ultrasound in twin pregnancy. *Ultrasound Obstet Gynecol*. 2016;47(2):247–263.
27. Giorgione V, D'Antonio F, Manji A, et al. Perinatal outcome of twin anemia-polycythemia sequence. *Ultrasound Obstet Gynecol*. 2021;58(6):813–823.
28. Nicholas L, Fischbein R, Aultman J, et al. Dispelling myths about antenatal TAPS. *J Clin Med*. 2019;8(7):977.
29. Tollenaar LSA, Lopriore E, Middeldorp JM, et al. Improved prediction of TAPS by delta MCA-PSV. *Ultrasound Obstet Gynecol*. 2019;53(6):788–793.
30. Tollenaar LSA, Slaghekke F, Middeldorp JM, et al. Prenatal staging of twin anemia-polycythemia sequence (TAPS) using delta MCA-PSV: a prospective cohort study. *Ultrasound Obstet Gynecol*. 2018;52(5):620–626.
31. Fortún Agud M, Marfil González M, et al. Multidisciplinary approach in TAPS diagnosis and treatment. *Life (Basel)*. 2024;14(9):1071.
32. Genova L, Slaghekke F, Klumper FJ, et al. Management of TAPS using IUT and PET. *Fetal Diagn Ther*. 2013;34(2):121–126.
33. D'Antonio F, Herrera M, Oronzii L, et al. Solomon technique vs selective fetoscopic laser photocoagulation for twin-twin transfusion syndrome: systematic review and meta-analysis of maternal and perinatal outcomes. *Ultrasound Obstet Gynecol*. 2022;60(6):731–738.
34. Tollenaar LSA, Slaghekke F, Middeldorp JM, et al. Fetal anemia in monochorionic twins: a review on diagnosis, management, and outcome. *Expert Rev Hematol*. 2023;16(1):9–16.
35. Slaghekke F, Pasman S, Veujoz M, et al. MCA-PSV to predict fetal hemoglobin levels in TAPS. *Ultrasound Obstet Gynecol*. 2015;46(4):432–436.
36. Slaghekke F, Favre R, Peeters SH, et al. Laser surgery as management option for TAPS. *Ultrasound Obstet Gynecol*. 2014;44(3):304–310.
37. Lopriore E, Hecher K. Spontaneous resolution of TAPS after laser therapy. *Am J Obstet Gynecol*. 2008;198(2):e4–e7.
38. van Klink JM, Slaghekke F. Neurodevelopmental outcome after Solomon trial. *Am J Obstet Gynecol*. 2016;214(1):113.e1–113.e7.
39. Klaritsch P. Complications in monochorionic twin pregnancies. *J Perinat Med*. 2025.