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Sonographic capture of acute exsanguination in a case of developing monochorionic cotwin demise

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Single fetal demise in monochorionic twin gestations represents a significant risk for cotwin demise and neurodevelopmental morbidity. The acute evolution of the demise of the cotwin is rarely witnessed. We report the acute hemodynamic changes documented during such an event that support the theory of a 'vascular sink' that can lead to consequences in the normal cotwin of a monochorionic twin gestation.

A 23-year-old gravida 3 para 2 (two previous term births) was referred to our center at 19+3 weeks' gestation for evaluation of twin–twin transfusion syndrome (TTTS). On the initial sonographic evaluation, Quintero Stage IV TTTS was confirmed, with evidence of severe cardiac dysfunction in the recipient twin.

Hemodynamic changes were observed during the examination, including sudden and rapid development of sustained bradycardia in the recipient twin with concomitant tachycardia and a marked increase in the middle cerebral artery peak systolic velocity (MCA-PSV) in the donor twin (Table 1). The patient was informed that these findings were suggestive of an impending recipient twin demise and elected not to undergo intervention. A final assessment, 90 min from the beginning of the examination, revealed a heart rate of 20 beats per min (bpm) in the recipient twin.

The patient returned to her referring physician who confirmed a dual fetal demise 24 h later. After induction of labor and delivery, the placenta underwent injection studies. Gross examination revealed one (donor to recipient) arteriovenous anastomosis and two venoarterial anastomoses (Figure 1).

Historically, the transfer of thromboemboli and thromboplastins from the demised fetus to the cotwin¹ is thought to be responsible for the demise or long-term sequelae in the cotwin. More recently, the theory of a 'vascular sink', created by a hemodynamic imbalance, has been proposed as the causative mechanism². Only indirect evidence supports this newer hypothesis because of the difficulty in capturing these dynamic changes *in utero* during an acute event. This includes demonstration of anemia in the surviving twin, either by direct determination of hematocrit at cordocentesis or indirectly by an elevated MCA-PSV in the surviving twin some time after the acute event has transpired³.

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Our case represents a ‘sonographic film’ of the acute hemodynamic changes. We witnessed a rapid increase in the MCA-PSV in the uncompromised twin fetus suggestive of acute anemia as well as reflex tachycardia suggesting a reflex response to hypovolemia. These changes were concomitant with the rapid development of sustained bradycardia in the cotwin.

This case adds further evidence to the hypothesis of the ‘vascular sink’, which occurs rapidly during the demise of the first twin of a monochorionic gestation. In addition, the acute onset of these changes seen in the current case suggests that interventions such as selective reduction (at a previable gestational age) or emergency delivery by Cesarean section (at a viable gestational age) will not prevent neurological injury in the cotwin.

Finally, a correlation between the type of vascular anastomoses and the risk of cotwin demise and development of pathological cerebral lesions has been proposed⁴, with an increased risk with superficial anastomoses such as arterioarterial or venovenous. However, the placental examination in our case illustrates that exsanguination can rapidly develop with any type and number of anastomoses.

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Figure 1.
Gross examination of the placenta following delivery, revealing an arteriovenous (AV) and two venoarterial (VA) anastomoses.

Table 1

Summary of hemodynamic changes at different time points during the examination

Hemodynamic measurement	Examination time		
	Start	50 min	90 min
Recipient			
FHR (bpm)	120	70	20
MCA-PSV (MoM)	1.2	—	—
Donor			
FHR (bpm)	140	160	180
MCA-PSV (MoM)	1.3	1.5	1.65

bpm, beats per min; FHR, fetal heart rate; MCA-PSV, middle cerebral artery peak systolic velocity; MoM, multiples of the median.