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Single-twin demise: Pregnancy outcome

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Single-twin demise can pose substantial risks for the surviving co-twin, including increased risk of fetal loss, preterm delivery, neurovascular injury, and end-organ damage. In this chapter, we summarise recently published research on the causes of single twin demise, the pathophysiology of injury to the surviving co-twin, and the evidence for current management strategies. The gestation at which single intrauterine fetal demise occurs, and the chorionicity of the multiple pregnancies, are the two most important factors when considering the risks to the surviving twin. Management should include fortnightly ultrasound scans for growth, umbilical artery Doppler studies, and liquor volume. In monochorionic twins, more complex Doppler assessment with middle cerebral artery Doppler velocimetry and a magnetic resonance imaging of the survivor's brain at least 3 weeks after single intrauterine fetal demise occurs should be carried out to look for evidence of neurological morbidity. With no other obstetric complications, dichorionic pregnancies can be delivered at term. Monochorionic pregnancies are more difficult to manage, and are often delivered between 34 and 36 weeks.

Introduction

Most multiple pregnancies are twin pregnancies (98%), and are associated with a higher risk of perinatal mortality compared with singleton pregnancies [1,2]. The overall incidence of single

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intrauterine fetal demise (sIUFD) after 20 weeks of pregnancy is variably reported between studies, but is estimated to be up to 6.2% of all twin pregnancies [3]. This complication may occur at any trimester, with potentially profound consequences. Some evidence shows that, in many cases, this occurs in the first trimester, sometimes even before the first ultrasound scan examination ('vanishing twin'), and this adds to defining the exact prevalence of this event. The introduction of National Institute of Health and Clinical Excellence guidelines relating to the care of multiple pregnancy in 2011 has placed emphasis on the requirement for first-trimester ultrasound examination to confirm viability, define chorionicity, and provide an opportunity for prenatal diagnosis [2]. In sIUFD, not only fetal mortality potentially increases in the surviving co-twin, but also the risk of perinatal morbidity, especially neurologic injury and risks associated with spontaneous and iatrogenic preterm birth and its complications. One of the key influential variables associated with the risk of co-twin morbidity and mortality is **chorionicity**. It is chorionicity rather than zygosity that influences the outcome of this twin complication, because of the placental angioarchitecture of intertwin circulations in monochorionic twins. Loss rates of up to 30–50% have been associated with monochorionic, monoamniotic pregnancies [4].

Aetiology

Fetal and maternal factors contributing to sIUFD are listed in Table 1. These include the presence of discordant structural congenital anomaly (with or without chromosomal differences) [5]. Unequal placental 'sharing' and selective intrauterine growth restriction (sIUGR) are 'sentinel associations', and velamentous cord insertion seems to compound risk. In monochorionic, monoamniotic twins umbilical cord 'entanglement' has classically been associated with risk of co-twin demise. Recent epidemiological studies indicate that the risk of this complication is more prevalent between 18 and 24 weeks (than the third trimester), and is often associated with co-existent pathologies, such as 'twin reverse arterial perfusion sequence [6]. Chorionicity, as described earlier, has an important bearing on the rate and outcome of sIUFD with monochorionic (irrespective of amniocity) pregnancies having a significantly higher rate compared with dichorionic pregnancies [7]. In a retrospective cohort study of twin pregnancies undergoing anatomic survey, McPherson et al. [8] found that monochorionic twins had an increased risk of sIUFD (OR 1.69, 95% CI 1.04 to 2.75) and a double demise (OR 2.11, 95% CI 1.02 to 4.37). Of all the double demises in the cohort, 70% occurred before 24 weeks [8]. The nearly universal 'intertwin' communicating placental circulation is responsible for many of the unique complications in monochorionic twins, including twin–twin transfusion syndrome (TTTS), twin anaemia–polycythaemia sequence (TAPS), and is the largest predisposing risk to monochorionic twins if sIUFD occurs. In TTTS, an unbalanced unidirectional arterio-venous shunt results in a net transfer of blood from one twin to another. The donor becomes hypovolaemic, which results in a decrease in the donors cardiac output and an increase in the peripheral vascular resistance. This induces tissue hypoxia, acidosis, and a rise in erythropoietin production [9,10]. The recipient twin is hypervolaemic with polyhydramnios, and has a significant risk of cardiac dysfunction, whereas the donor twin is hypovolaemic with oligohydramnios. Both twins are at high risk of intrauterine death. In contrast to TTTS, which is essentially amniotic fluid discordance, TAPS is characterised by a severe haemoglobin discordance (at least 8g/dL)

Table 1
Reasons for single intrauterine fetal demise.

Fetal
Infection
Chromosomal anomaly
Structural anomaly
Cord anomaly (e.g. entanglement and velamentous)
Placental (e.g. twin–twin transfusion syndrome, twin anaemic polycythaemia sequence, and selective intrauterine growth retardation)
Maternal
Hypertensive disorders (i.e. Pre-Eclampsia)
Thrombophilia, abruption
Iatrogenic
Selective feticide
Complication after laser therapy for twin–twin transfusion syndrome