Vasa Praevia: a descriptive review of existing literature and the evolving role of ultrasound in prenatal screening

Abstract

Introduction: Literature addressing the feasibility of prenatal detection of vasa praevia during the mid-trimester morphology ultrasound scan is scarce, as is a lack of consensus about the appropriate management of pregnancies once it is detected.

Method: The following descriptive review will provide historical context about the clinical significance, epidemiology, diagnosis and outcomes of pregnancies complicated by vasa praevia. It will also examine the role of ultrasound in the diagnosis of vasa praevia, and will examine current evidence surrounding this debate of whether routine screening for vasa praevia is possible, beneficial, or cost-effective.

Conclusion: Finally, it will highlight the need for increased research into effective management of pregnancies at high risk of, or affected by vasa praevia to reduce fetal mortality and maternal and fetal morbidity associated with the condition.

Keywords: antenatal, bilobed placenta, colour Doppler, placental complications, screening, ultrasound, vasa praevia, velamentous umbilical cord insertion.

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Introduction

Vasa praevia (VP) is an uncommon obstetric condition that has a perinatal mortality rate of approximately 60% if not prenatally detected and appropriately managed.[1] It occurs when exposed fetal vessels traverse the amniotic membranes between the baby's presenting part and the internal cervical os.[2] The term 'vasa praevia' is derived from the Latin words 'vasa', meaning vessels, 'pre' or 'prae' meaning before and 'via' meaning way. Hence fetal vessels lie before the baby and in the way of the birth canal. This condition often results in fetal death when the membranes rupture either spontaneously or artificially, leading to rapid fetal exsanguination. Because VP is rare, it is extremely difficult to conduct prospective studies to record the incidence of the condition, and despite improvements in technology, VP is still under-reported or miscoded in maternal morbidity and mortality coding systems and epidemiological data collections.[1,3] Nonetheless, the incidence of VP has been estimated at 1 in 2500 births, although has been reported to vary between 1:513 and 1:6000 in naturally conceived pregnancies, and as high as 1:293 in IVF-assisted pregnancies.[4–6] The most recent published report by Hasegawa, *et al.*[7] found an incidence of 1:365 (10 cases of VP in 3647 pregnancies screened between 2006 and 2011) however the method of conception is not identified, presumably a mix of naturally and artificially conceived pregnancies.

There are two main types of VP, Type I with velamentous insertion (VCI) of the umbilical cord and Type II with bilobed or succenturiate placenta.[8] In a velamentous insertion, the cord inserts into the membranes through which unprotected vessels then run until they end in the placenta. In Type II VP, exposed vessels run through the membranes between lobes of a bi-lobed placenta.[3] VCI occurs in up to 1:100 singleton pregnancies (higher incidence in multiple pregnancies) and when located in the upper uterine segment, rarely causes complications.[9] However, if the VCI occurs in the proximity of the internal os, below the baby's presenting part, it becomes a VP.[2]

Vasa praevia, whether Type I or Type II, is thought to arise from a placenta that covers the cervix in the early part of pregnancy. As the pregnancy progresses, because the region over the cervix is poorly vascularised, the placenta grows preferentially toward the upper part or fundus of the uterus. At the same time, the placental tissue overlying the cervix undergoes atrophy, leaving exposed vessels running through the membranes.[8]

In itself, VP is not dangerous to the mother, although when accompanied by placenta praevia there is an additional risk of maternal haemorrhage.[2] The danger to the fetus from VP is due to exsanguination when the exposed fetal vessels rupture when the amniotic membranes rupture, either naturally or via amniotomy.[10] Compression of these vessels by the fetal presenting part may also place the fetus in jeopardy.

Risk factors

Risk factors for developing VP include a low-lying placenta during the second trimester, multi-fetal pregnancies, IVF-assisted pregnancies, pregnancy involving multi-lobed placentas and velamentous insertion of the cord.[2,6,11–13] Evidence suggests that the highest risk for VP is the presence of placenta praevia at the second trimester ultrasound scan. In a comparison of women with VP and a control group of women with uncomplicated pregnancies, Francois, *et al.*[12] reported 69.2% of VP cases had a placenta praevia observed during the second trimester scan, compared to 3.8% in the control group (OR = 56.3, P < 0.000001). Oyelese, *et al.*[1] observed a similar association through a retrospective study showing 62.1% of VP cases had either a low-lying placenta or placenta praevia identified during the second trimester scan. Recently, a link has also been suggested between cord insertions into the lower uterine segment identified in the first trimester (9 to 13 weeks*)* and the development of VP.[5,7,14–16]

Methods

Search strategy

The search strategy used for the review incorporated a search of literature published since 1980 using a number of databases included Medline, Proquest, ScienceDirect, EBSCO, Wiley Interscience, EMBASE, CINAHL and the Cochrane database. The search terms 'vasa previa', 'vasa praevia', 'velamentous', 'bilobed placenta' and 'succenturiate placenta' were used. The websites of the International Vasa Previa Foundation and the UK Vasa Praevia Raising Awareness organisation were also reviewed for links to literature that may not have been indexed in medical databases. Articles addressing the antenatal diagnosis of VP by ultrasound, amnioscopy or MRI were included if published after 1980 in English. The literature included case reports and series, retrospective studies (both case-control and case series), prospective studies, clinical guidelines and literature reviews.

Data review

The initial search strategy was conducted in 2010 and yielded 954 articles. A review of the article titles for relevancy reduced the number to 126. Further examination of the abstracts and use of the date and language exclusion criteria resulted in a final total of 107 articles for review. Updated searches were conducted in February and November 2012 which led to a further 24 relevant documents being identified, resulting in 131 articles for review. Many of these were single case reports that were often 'accidental' diagnoses of VP or cases of fetal demise due to unidentified VP. While useful when considering the seriousness of the condition and its 'asymptomatic' nature, these types of case reports did not add to the body of knowledge regarding diagnosis and therefore not all have been included, leaving 75 relevant articles or guidelines reviewed.

Prenatal diagnosis

Diagnosis utilising ultrasound

The earliest reports of the ability to detect VP using ultrasound appeared in the late 80s and early 90s.[17–20] Successful identification of VCI however, was questioned due to low observed detection rates,[21,22] leading to the suggestion that improvements in accuracy of scanning, including detection of VCI, could be enhanced by "the use of colour flow Doppler imaging".[21] Nonetheless, later studies examining placental cord insertion using ultrasound demonstrated significantly better results in detecting VCI, possibly due to improvements in ultrasound technology or the skill of the sonographers.[23–25] These studies were important in demonstrating that visualisation of the cord insertion site and identification of abnormal insertions was possible by ultrasound. The following years saw an increase in published studies focused on antenatal diagnosis utilising various ultrasound imaging techniques,[8,26,27] with additional case reports showing that advances in ultrasonography, such as 3- and 4-dimensional ultrasound, especially when combined with colour and power Doppler angiography resulted in more accurate diagnosis of VP and in particular, the ability to accurately map out the location of the vessels prior to caesarean section (CS) delivery.[28–30]

Timing of ultrasound diagnosis

Since the first report by Gianopoulos, *et al.*[17] in 1987 there have been numerous published case reports of sonographic diagnosis of VP (both 2D and 3D with and without colour and power Doppler) resulting in good perinatal outcomes.[19,28,29,31–47] Timing of ultrasound scans in the larger studies and case reports varied, ranging from late in the first trimester to term.[5,27]

Two studies published by the Showa University School of Medicine in Tokyo examined detection of the cord insertion in the late first trimester and during the routine mid-trimester scan at 18–20 weeks.[15,48] These investigators found that identification of the placental cord insertion was possible in 93.5% of cases at the time of the nuchal scan (9–11 weeks). They also found a considerable risk for complications later in pregnancy in those cases in which the cord inserted into the lower third of the uterus in the first trimester. This included a relative risk of 9.32 for VP and 11.48 for a low-lying placenta at term.[15] A further study at the same centre confirmed the link between a low cord insertion identified between 9 and 13 weeks' gestation and the development of placental abnormalities such as placenta praevia, velamentous insertion and VP, independent of the relationship between the insertion site and the placenta.[16]

Sepulveda, *et al.*[9] undertook a prospective year-long study and screened 533 consecutive pregnancies during the nuchal translucency scan (11–14 weeks) for VCI. The placental cord insertion was visualised in 100% of cases with the identification of five cases of VCI (incidence of 0.9%). These cases were then monitored at the second trimester scan, and again at delivery. They concluded that during the twelve-month study period, no cases of VCI were missed. These authors suggested that the ease of diagnosing VCI at the nuchal scan in the first trimester may be better than at the time of second trimester scan. Early identification of VCI does enable early management and closer monitoring of suspicious observations, however scanning for VP during the first trimester will not identify all cases due to the changing position and structure of the placenta as described previously. Furthermore, there may be more false positive diagnoses in the first trimester. Bilobed, succenturiate placentas and velamentous insertions may not yet have developed during the first trimester. While it may be beneficial to identify cases of cord insertion into the lower third of the uterus that potentially may develop into VP later in pregnancy, it would be premature

to rely purely on identification at this time and not additionally screen during the second trimester. While the numbers of women undertaking a nuchal screen are increasing, there are still greater numbers attending for the mid-trimester morphology scan than the initial nuchal scan. Consideration also needs to be given to the psychological impact on women when diagnosed so early in the pregnancy with an abnormality that may 'right itself' as the pregnancy progresses and thus prove to be relatively benign. The evidence from all the reviewed case-reports and studies indicated the most reliable timing for prenatal diagnosis of risk factors for both Types I and II VP is during the second trimester.

It is standard, at the present time, to routinely identify both the fetal cord insertion and the placental location as part of the mid-trimester morphology scan, but it is not yet standard practice to identify the placental cord insertion. However, it has been demonstrated that routine identification of placental cord insertion at the time of the second trimester morphology scan can be easily and consistently accomplished, and on average, takes less than one minute.[25,27] Thus, it is logical that screening for VP in women with the identified risk factors (including low-lying placenta, IVF-assisted conception, multi-fetal pregnancy and multiple-lobed placentas) is conducted at this scan rather than in a separate screening scan. Diagnosis at 18-20 weeks still allows sufficient time for additional monitoring of fetal wellbeing as well as a planned CS. A diagnostic algorithm proposed by Derbala, *et al*. provides useful guidance for when further ultrasound screening for VP is warranted.[49]

While ultrasound has been suggested to be the most effective diagnostic tool for antenatal diagnosis of VP, some authors have also suggested that Magnetic Resonance Imaging (MRI) may be useful.[50–53] While there may be some recognised benefits of MRI in providing additional information to both grey-scale and colour Doppler sonography such as greater detail of vessel location prior to CS and further confirmation of the placental forms (e.g. single, placenta praevia, bilobed, succenturiate lobe) when used during the third trimester,[50,52] the cost and extra time required is generally prohibitive.

Issues preventing prenatal diagnosis

Mounting evidence suggests that risk factors for VP can be identified by the time of the routine second trimester morphology scan, yet there is still an apparent lack of consensus in the obstetric community as to whether routine prenatal screening for VP is actually feasible, and there is little reliable data available to indicate whether it is being achieved. A recent survey of members of the Society for Maternal-Fetal Medicine in the United States revealed that only 43% of maternal-fetal medicine specialists had a protocol for screening.[54] The results of a survey sent to all obstetric and fetal-maternal consultants across England and Wales in 2006[55] were also significant in demonstrating that 30% of respondents could not name a single risk factor for VP and only 33% were offering transvaginal colour Doppler scans for identification of fetal vessels over the cervical os. However, since the report was published, the Royal College of Obstetricians and Gynaecologists (RCOG) in the UK and the Society of Obstetricians and Gynaecologists of Canada have published guidelines for the diagnosis and management of VP. The guidelines do not recommend universal screening;

however they do include the recommendation for evaluating the placental cord insertion in women with identified risk factors for VP during the second trimester scan.[4,56] It is disappointing that many other countries, including Australia, New Zealand, and the United States of America, have not followed suit and implemented practice guidelines for both the diagnosis and management of VP. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists released a College Statement in July 2012 that does not address the management of VP but recommends targeted screening with ultrasound for all women with risk factors and the identification of the placental cord insertion site for all singleton pregnancies.[57] While prenatal diagnosis of vasa praevia by ultrasound may not be possible in every case (factors such as obesity, fetal position and abdominal scarring may interfere with identification of the cord insertion or fetal vessels near the os) the literature does indicate it is feasible in the majority of cases during the late first and second trimesters, and at little or no increased demands in personnel, equipment and time.

Management of diagnosed VP pregnancies

The relative rarity of VP and the severe morbidity and mortality associated with the condition, makes it difficult to conduct trials evaluating the appropriate management of affected pregnancies. Clearly, delivery by caesarean should be performed prior to rupture of membranes to avoid rupture of the exposed fetal vessels and fetal exsanguination. However, there is limited evidence available to determine the ideal timing of delivery, or whether expectant management is entirely appropriate.[58] Nonetheless, it is certain that if delivery is done after the membranes rupture, the fetal prognosis is poor.[1]

Historically, when VP was suspected during labour (in the presence of bleeding and/or fetal heart decelerations) the suggested management was the immediate delivery of the baby, by caesarean delivery followed by aggressive resuscitative measures of the newborn including transfusion of blood or blood products.[59–71] Despite these measures, the rate of morbidity and mortality following undiagnosed VP remains high, with the largest study showing the rate of perinatal mortality in prenatally undiagnosed cases to be 56% compared to 2% in those prenatally diagnosed.[1]

Studies have suggested the optimum timing for CS delivery (with diagnosed VP) is approximately 34–35 weeks without amniocentesis to reduce the risk of membrane rupture while also balancing iatrogenic morbidity due to premature delivery.[1,72] Oyelese, *et al*.[1] recommended early hospitalisation at about 32 weeks of gestation, and administration of corticosteroids to promote fetal lung maturation. Early hospitalisation was rationalised as a way to facilitate immediate access to an operating theatre, and the possibility of emergent caesarean delivery in case of rupture of the membranes and/or vessels. The benefits of early hospitalisation and regular fetal heart rate assessment were highlighted in the study by Kanda, *et al*.[5] with four of their ten cases of VP demonstrating non-reassuring fetal heart rate during preterm labour. The authors suggested this may have been due to compression of the velamentous vessels. However, no one has studied the option of outpatient management as an alternative in selected cases where there is no

bleeding or evidence of preterm labour, and where the patient is deemed at low risk for preterm rupture of membranes based on a long closed cervix on transvaginal ultrasound and a negative fetal fibronectin test.

A 20 year retrospective review conducted in Israel indicated that of the 19 cases of VP diagnosed during that time, five cases required urgent delivery (between 33–36 weeks) due to premature labour, rupture of membranes or vaginal bleeding.[73] All prenatally diagnosed cases had been scheduled for a CS delivery at 35–36 weeks. The authors also highlighted the risk of delaying delivery after prenatal diagnosis due to the risk of premature rupture or spontaneous onset of labour, while also being conscious of delivering too early and increasing the likelihood of complications due to prematurity.

Another retrospective review of 13 prenatally diagnosed cases of VP, was carried out by Romero, *et al*. between 2007 and 2010.[74] Gestational age at delivery varied between 24 and 38 weeks with the majority of deliveries occurring at 34 and 35 weeks. Although there was only one neonatal death (at 24 weeks due to extreme prematurity) there was significant iatrogenic morbidity associated with late preterm delivery, including over 40% of babies having respiratory distress syndrome (RDS). It was noted that eight of the mothers had received Betamethasone for fetal lung maturity, however the authors did not indicate whether there was a correlation between non-administration of Betamethasone and RDS or whether the women were hospitalised or managed as outpatients.

Robinson and Grobman[72] have developed a decision analytical model to evaluate the optimum timing of delivery of prenatally diagnosed cases of VP. They compared 11 different strategies for timing of delivery, including a combination of gestational age, administration of corticosteroids and amniocentesis to assess fetal lung maturity. The model concluded that the best outcome was achieved when delivery occurred at 34 weeks gestation after administration of antenatal corticosteroids but without amniocentesis.

In their population-based study of 246, 525 pregnancies in Israel, Weintraub, *et al*.[13] identified 237 cases of VP. Multivariate analysis identified that the risk of premature rupture of membranes was greater in cases of VP (OR = 1.8, CI = 1.2–2.6, P = 0.002) as was intrauterine growth restriction (OR = 4.3, CI = 2.7–6.9, P < 0.001) and placental abruption (OR = 8.2, CI = 4.8–14.1, P < 0.001) when compared to women without VP. Early hospitalisation and monitoring of women diagnosed with VP would allow earlier recognition of problems and enable timely response to emergent complications.

Evidence from the studies reviewed, taken together, highlight that while prenatal diagnosis of VP may go a long way to decrease perinatal mortality, there is still the chance of significant neonatal morbidity if diagnosed women are not appropriately managed.

Global perspectives on diagnosis and management of VP

While it is clear that planned caesarean in cases of vasa praevia leads to better outcomes, surveys conducted in both the UK and the USA indicated that only a minority of clinicians offer hospitalisation prior to late preterm elective delivery and even fewer offer steroids for fetal maturation.[54,55] Thus, there does seems to be little consensus on the best management plan, with only the UK and Canada having published practice guidelines.[4,56] While both of these guidelines acknowledge that VP can be diagnosed in most cases during the second trimester fetal anomaly ultrasound scan, neither of them recommend universal screening for VP. A review by the UK National Screening Committee indicated that one of the reasons universal screening of all pregnant women for VP was not recommended was due to the lack of agreed management guidelines for pregnancies once diagnosed.[75] Both of these clinical guidelines recommend consideration of early hospitalisation (28–32 weeks), administration of corticosteroids and CS delivery prior to the onset of labour, although do not specify a recommended gestation. The lack of evidence for management of VP following diagnosis indicates an urgent need for further research to establish the required evidence-base.

Discussion and conclusion

The evidence presented in the existent literature on VP suggests that the use of screening for VP during the second-trimester morphology scan will improve fetal outcomes in those cases diagnosed. There are few other conditions in obstetrics where prenatal diagnosis and appropriate management makes such a difference between survival (97% in diagnosed cases versus 44% in cases not diagnosed) and death. Visualisation of the placental cord insertion has been found to be achievable with little impact on the time taken for the scan, no need for extra personnel or equipment, and with a high degree of accuracy. Incorporating this with the additional knowledge of the known risk factors for VP, including low-lying placenta, low cord insertion in the first trimester, velamentous cord insertion, IVF and multi-fetal pregnancies makes the likelihood of detecting VP prenatally even higher. While diagnosis is possible in the first-trimester, there seems to be little benefit from incorporating screening as part of the nuchal translucency scan, as detection is more likely to be missed at this point (particularly for Type II VP) and management protocols and outcomes are not likely to be affected. With most women undergoing a mid-trimester morphology scan, there seems to be little argument (based on evidence) against screening for VP risk factors as part of this scan. The literature is convincing that prenatal detection using ultrasound does make a significant impact on perinatal outcomes, with a reduction in mortality rates. The outdated belief that morbidity and mortality from VP is unavoidable can no longer be supported.

While diagnosis of VP is improving and technology is enabling earlier and easier detection, there remains a need for further evidence to establish the optimum management for these pregnancies to provide the best outcome for both mother and baby. Current recommendations in the literature seem intuitive; however, they lack the evidence base required for rigorous guidelines. With rates of Artificial Reproductive Technologies (ART) and CS on the increase, the rise of incidence of VP is likely. The need for a multi-centre, collaborative study is critical to yield reliable data on incidence, prenatal diagnosis rates, management and outcomes for Australian women and their babies.

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