

The clinical implications of single umbilical artery: A case report and multifaceted review

B McCully^a, S H Dodampahala^a, A S Menon^b, N D'Sa^b, E Day^b, M Ahmed^b

Abstract

A single umbilical artery (SUA) is a notable anomaly in fetal development, characterized by the absence of one umbilical artery. This condition, detectable through routine ultrasound screening as early as the 12th week of gestation, raises significant obstetric and perinatal considerations. Despite its prevalence, the pathogenesis of SUA remains largely idiopathic, potentially indicating intrinsic anomalies that may be associated with chromosomal and congenital abnormalities in up to 50% of cases³. This paper aims to discuss the nature of SUA, including its historical context, diagnostic approach, evolutionary significance, and implications for antenatal care, focusing on optimizing outcomes through a collaborative, patient-centric care model.

Introduction

The normal foetal umbilical cord consists of two arteries and one vein. The most common abnormality occurs when one of the umbilical arteries is absent (SUA), leaving a single vessel with a single vein. The left artery is more commonly absent than the right¹. SUA can be antenatally diagnosed through routine ultrasound screening as early as 12 weeks of gestation. It is one of the most common foetal abnormalities and may be associated with a range of adverse outcomes, including stillbirth, intrauterine growth restriction (IUGR) and

low birth weight. The pathogenesis is uncertain and, in many cases, is idiopathic. It may, however, reflect intrinsic anomalies that result in either primary agenesis or secondary atrophy during the second trimester of gestation⁴. Such an aetiology may explain why chromosomal and congenital anomalies are found in up to 50% of foetuses with antenatally diagnosed SUA. Two-thirds will have Trisomy 18, while other congenital malformations will include cardiac, neurological, genitourinary and gastrointestinal dysfunction. Because of this, SUA is associated with increased obstetric and perinatal risk.

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^a Professor/Consultant Monash Medical School and Consultant MBPH Vic 3500

^b Monash Medical School Vic 3500

Correspondence: SHD, e-mail: senani.dodampahala@monash.edu



<https://orcid.org/0000-0002-6220-7723>

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Case report

A 26-year-old pregnant woman was found to have a two-vessel cord at the time of routine morphology scanning at 20 weeks of gestation. Previously, in 2021, she had had an unexpected stillbirth diagnosed in late pregnancy. She had no risk factors for adverse outcomes and had an otherwise normal antenatal history. Labour was induced and was complicated by cervical dystocia at 3cm. She had a lower uterine segment caesarean section (LUSCS), which birthed a baby that was large for gestational age, weighing 4470g. Blood sugars were normal, and routine screening failed to identify any attributing factors that may have led to foetal demise.

In the current pregnancy, the woman's body mass index (BMI) was 34; she had a healthy lifestyle and did not smoke or drink alcohol. Her routine first-trimester antenatal investigations for viral serology, including hepatitis B and C, HIV, and varicella, were all normal. She tested negative for syphilis and was rubella immune with no positive blood group antibodies. She had a low risk for aneuploidy using first-trimester combined screening. Her 20-week scan was unremarkable, showing a morphologically normal foetus consistent with dates and a normal, non-*praevia* placenta. Amniotic fluid volume was normal, and the cervix was long and closed, measuring greater than 25 mm. A 2 vessel cord was confirmed with a single umbilical artery demonstrated at the level of the foetal bladder. Appointments for regular antenatal attendance were recommended. Unfortunately, the woman was unable to comply with many of these. English was a second language, and though her care was provided through a public hospital system, socio-economic and cultural constraints made it difficult for her to engage. Consequently, despite attempts to reach out and facilitate additional oversight, the woman was seen sporadically. When care was attended, opportunistic bedside surveillance was used to assess foetal growth and wellbeing.

She presented to the birthing suite in spontaneous labour at 40 weeks with ruptured membranes. The liquor had thick meconium staining. The foetal heart rate was auscultated between 140-145 BPM. She declined vaginal examination, CTG monitoring and IV cannulation. Despite these concerns, the baby was born 2 hours later in good condition with APGARs 9 at 1 and 5 minutes. The baby weighed 2450 kg and was vigorous with simple tactile stimulation. The third stage was completed in 15 minutes. The placenta and

membranes were delivered complete and the inspection of the cord confirmed a single umbilical artery. Arterial cord blood showed a pH of 7.1 and a lactate of 9.5. The venous cord blood had a pH of 7.2 and lactate 9.8. A moderate atonic post-partum haemorrhage (PPH) of 800mL ensued and was resolved with an intra-muscular injection of a Syntocinon 10 units. The mother's full blood count after birth was normal.

The baby was admitted to the special care nursery to observe and monitor acidosis, blood glucose and respiratory effort. No active intervention was required. The mother sought to be discharged the next morning and went home safely with her baby. A neonatal follow-up was arranged for renal ultrasound and echocardiography two weeks later. Histopathology of the placenta confirmed a 2-vessel cord with evidence of widespread high-grade villitis and placental infarction with foetal-vascular malperfusion.

Discussion

The normal human umbilical cord contains two umbilical arteries and one vein, facilitating the bidirectional exchange of nutrients and waste between the fetus and placenta⁵. A single umbilical artery (SUA) is a condition in pregnancy where one of the two umbilical arteries is missing. The first documentation of a single umbilical artery (SUA) dates back to the 18th century⁴. The condition was initially described by the French obstetrician and anatomist Jean-Louis Baudelocque (1745-1810) in 1774. He was a prominent French obstetrician best known for his work, "*L'Art des accouchements*" (The Art of Child Delivery), which critically described the shape and size of a mother's pelvis as a predictor of disproportion at birth. Though he failed to understand its significance, Monsieur Baudelocque's description of SUA was significant because it highlighted a deviation from the normal anatomy of the umbilical cord. This variation is now recognized as one of the most common malformations of the umbilical cord, occurring in about 0.5% to 1% of singleton pregnancies and somewhat more frequently in multiple pregnancies, particularly those of monozygotic twins⁶.

Diagnosis

Through conventional 2D ultrasound imaging, the umbilical vessels can be identified as early as the first trimester of gestation. SUA is most commonly, however, identified as part of the foetal anatomic survey

during the second trimester². Colour flow Doppler allows visualization of the vessels as they run alongside the foetal bladder, most clearly seen at the cord's insertion to the foetal abdomen (Figure 1). In cases of SUA, ultrasound shows only a single artery with the vein (Figure 2). The left artery is often absent;

however, most studies indicate that laterality is insignificant (Figure 3). Confirmation of SUA is made postnatally through gross and histological examination of the umbilical cord, documenting the presence of a single artery accompanying the vein (Figures 4, 5, 6).



Figure 1. Normal colour flow Doppler ultrasound of umbilical vasculature around the bladder.



Figure 2. Ultrasound of cross section of the umbilical cord showing SUA and vein.



Figure 3. Colour flow Doppler ultrasound.



Figure 4.

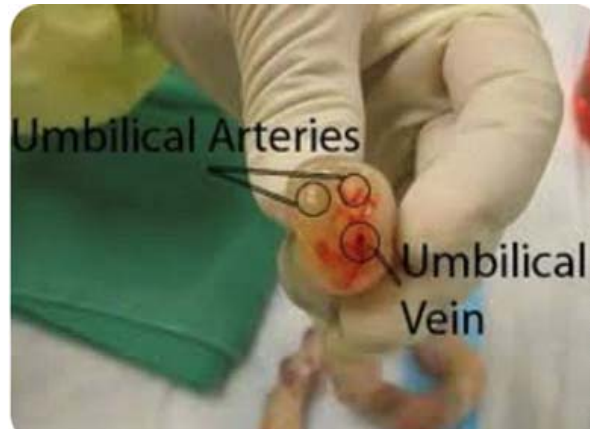


Figure 5.

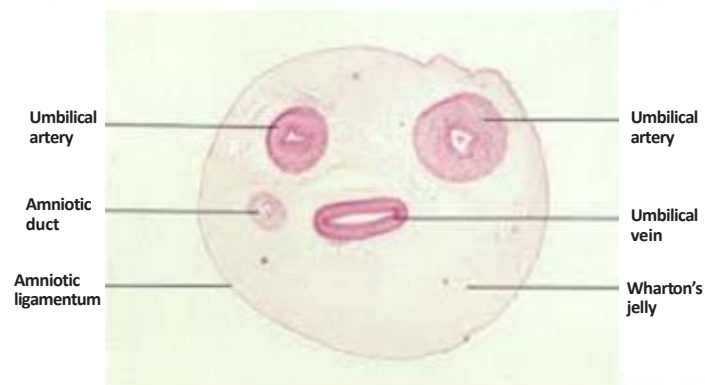


Figure 6.



Figure 7.

Figure 4, 5, 6, 7. Naked eye visualization and cross section of a normal umbilical cord consisting of two umbilical arteries and one umbilical vein.

Normal development

Umbilical cord development begins at week 3 of embryological development. At that time, the embryo is a trilaminar disc attached to the decidua basalis by a connecting stalk, which forms the primitive umbilical cord. The embryo folds during week four^{8,4}. This creates a tube that closes ventrally. The connecting stalk attaches to this surface and includes the yolk sac, which forms the omphalomesenteric or vitelline duct, and the allantois, an outpouching of the endodermal hindgut. By term, both will have typically been involuted. Between the fourth and eighth weeks, there is an increase in amniotic fluid, which expands the amniotic cavity to fill the chorionic space and causes elongation of the connecting stalk, which is now lined by the amnion⁸.

Starting in week three, endothelial precursor cells in the mesoderm surrounding the allantois coalesce to form small capillaries. Vasculogenesis continues, and by the end of the third week, the capillaries have grown to establish a functional vascular network within the connecting stalk. During the same period, the arterial and venous systems within the embryo are developing. The arterial system is initially established as the paired dorsal aortae. Early in the fourth week, umbilical arteries arise from either side from branches that later form the internal iliac arteries. The foetal umbilical arteries ascend the abdominal wall and anastomose with the umbilical cord's vascular network⁸.

Similarly, the umbilical veins are also originally bilateral. In the second month, the right umbilical vein regresses, leaving the left umbilical vein to enter the foetus and connect to the ductus venosus of the developing liver. With the initiation of fetal heart activity during week four, the umbilical arteries carry deoxygenated blood to the placenta, and the umbilical vein carries oxygenated blood back to the fetus from the placenta⁹.

During week seven, the growth of the foetal intestines causes them to herniate into the umbilical cord, leading to the cord's elongation. Between weeks ten and twelve, the intestines return, and the extraembryonic mesoderm of the cord develops a rich extracellular matrix known as Wharton's jelly, which helps to protect the vessels from compression¹⁰. The umbilical cord continues to elongate during the second trimester, allowing the fetus ample space for movement and growth.

The cord typically measures between 50 and 60 cm at birth and is 2cm in diameter. Unlike other vascular

systems, the umbilical vessels within the cord do not branch. This is unique and can be understood from an evolutionary perspective. Unlike other systems where branching is advantageous to enhance supply and flow distribution, the cord vessels are designed solely to ensure rapid exchange between the fetus and placenta with minimal distraction or impediment. The vessels are, however, coiled with up to 40 helical turns. (Figure 7). These are pivotal in providing resilience to withstand cord compression and torsion. This is vital as the fetus moves and grows within the womb. Additionally, the helical structure facilitates greater elasticity and flexibility, allowing elongation and movement of the cord as the fetus develops and moves. This may reduce the risk of knots and kinks or of becoming tangled, which could lead to compromised blood flow. The turns may also regulate blood pressure within the umbilical cord vessels, creating a less turbulent and more consistent laminar flow that can better withstand transient changes in the intra-uterine environment¹¹.

After birth, the arteries of the cord contract, preventing afferent loss from the baby's circulation. The remnants of the umbilical arteries within the baby become the medial umbilical ligaments, found on the anterior abdominal wall, running from the umbilicus inferiorly to the pelvis. The remnant of the umbilical vein becomes the ligamentum teres hepatis, which extends superiorly from the umbilicus to connect to the falciform ligament of the liver.

Of mice and men – evolutionary trends

In the context of this discussion, it is interesting to note that a single umbilical artery (SUA) is normal in a wide range of species, from cetaceans (whales, dolphins, and porpoises) to some members of the ungulates (hooved animals), including domestic horses, zebras and Camels⁴. Rhinoceroses, bats and many reptiles also have a single umbilical artery as part of normal physiological development. In humans and other primates, two umbilical arteries may represent an adaptive change from this pattern. This suggests an evolutionary improvement with specific advantages that support a more efficient placental blood supply, possibly in response to the demands of foetal development. A particularly compelling argument for this evolutionary shift is its potential connection to brain development. The human brain is notably large and complex compared to other mammals, requiring significant nutritional and oxygen support during fetal development. The evolution of a second umbilical artery could be an adaptation that emerged to meet these

demands, providing more robust support for critical brain growth and development and enabling higher brain functions, including cognition, language, and social behaviours. While this sounds persuasive, we must remember that the trend towards two umbilical arteries is also seen in cats and dogs, cows and other animals of pasture such as sheep and goats, and, yes, less loftily, mice and other rodents⁴.

Despite such mixed company, it may still be reasonable to argue that the persistence of a SUA in human pregnancies may represent a failure or aberration of normal embryology. It may arise as a programmed failure of primary development or as a secondary atrophy of vasculogenesis and, as such, may herald an association with other congenital or chromosomal abnormalities such as trisomy 13, 18, and 21. A retrospective study in 2014 reported that malformations were found in 17.6 per cent of SUA pregnancies. In 2020, Ebbing et al.⁹ cited a lower prevalence of 11%. Public health information from Safer Care Victoria suggests that the rate may be even higher, with 50% of SUA cases reportedly associated with antenatal malformations. This is collaborated by other studies that report a rate between 13 to 56%. Autopsy studies by Rittler et al. found that SUA increased the risk of malformations by 3 to 9 times, with urinary and gastrointestinal malformations showing the strongest links. Additionally, Vasanthalakshmi et al. reported that of 59 cases of SUA in 6711 singleton pregnancies, 22 had anomalies that were either cardiac or urogenital. The former included ventricular and atrial septal defects and hypoplastic right heart syndrome. Gastrointestinal and urogenital complications included hypospadias, unilateral renal agenesis and imperforate anus¹⁰. Paediatric complications include Vesicoureteral reflux (VUR), which occurs in 4% of isolated SUA cases. VUR causes retrograde urine flow from the bladder to the upper urinary tract, increasing the likelihood of UTIs and pyelonephritis. The incidence of urinary tract infections (UTIs) in SUA cases, even without identifiable genitourinary abnormalities, remains about three times higher (1.9%) compared to non-affected pregnancies (0.6%). This risk remains until late childhood.

What else is going on?

A retrospective study by Jerzy Stanek revealed that intra-uterine foetal growth restriction was found in 29 per cent of pregnancies affected by SUA. This is supported by other retrospective cohort studies reporting a higher risk for IUGR as well as an increased

risk for preterm deliveries before 34 weeks of gestation. A 2021 cross-sectional study furthers this notion, also reporting a higher prevalence of intrauterine foetal death, early neonatal death, and preterm birth in SUA cases⁵. This may be attributed to the sequela of congenital morbidity or chromosomal disjunction as described earlier, but may also reflect the impact of environmental aetiological factors such as smoking and drug exposure as well as maternal health conditions including diabetes and hypertension, which may themselves be implicated as causal with placental dysfunction and growth impairment⁵. However, let us consider specifically the impact of a single umbilical artery, how this may affect placental function in human pregnancy and, in particular, how this may be less advantageous for normal growth and well-being.

Fractals – friend or foe

Fractal geometry stems from the Latin fractus, which means broken. It describes shapes of any size characterized by a self-similar structure regardless of scale. Benoit Mandelbrot, a pioneer in this field, described fractals as shapes composed of smaller parts, each similar to the whole. This concept is found in naturally occurring phenomena such as mountains and trees where, for example, parts of the whole, such as a rock or a twig, resemble the pattern of the larger form. This property is called scale invariance. It demonstrates a tendency to preserve patterns that work well¹¹. The principles parallel the branching patterns of biological supply networks such as the pulmonary airways and the vascular system. Each demonstrates a recursive, self-similar pattern of branching that is evident across multiple scales of size within the organism. It highlights an evolutionary trend to preserve systems or designs with the greatest potential to withstand redundancy and optimize the cost-effective flow of fluids through a supply system, whether air or blood, to perfuse individual cells¹¹. This is called Murray's law, and it postulates that the geometry of vascular branching will utilize the best design for efficiency.

The fractal imperatives of vascular development suggest that capillary networks within the placenta will form predictably. They will eventually arise as the terminal vessels of larger afferents that branch and divide as they move distally from the cord into the placenta. This branching pattern leads to a fixed perfusion area for each vessel. In the case of a single umbilical artery¹¹, if the placental volume is preserved, the constraints of fractal design would task each

capillary to journey further through the tissue to compensate for the lack of a second vessel. Thus, encumbered, perfusion would be less effective, and the placenta would be less able to meet the needs of the growing foetus, which may explain why SUA is associated with a higher risk of growth restriction, foetal distress and other pregnancy complications, including preeclampsia. Indeed, while this may be true, it is insufficient to account for the entirety of change or adaptation seen in pregnancies affected by SUA and thus may not fully account for the variations of outcome encountered. A second serving is at hand.

“Please, sir, I want more,” Oliver Twist

Fractal design and limitation may be true for organs such as the liver, but others, including skeletal muscle and placenta, can increase capillary density beyond the limits imposed by predicted geometry using local chemical mediators to regulate the growth and proliferation of capillaries in tissues to meet local demand. An example of this occurs in response to regular exercise when capillary networks expand in active muscles to provide the required oxygen and support¹¹. Tissue growth factors are pivotal in inducing and regulating capillary supply networks. Angiogenesis describes endothelial cells’ proliferation, migration, and differentiation to form new capillaries from pre-existing blood vessels. Vascular Endothelial Growth Factor (VEGF) is a prime example. It affects physiological processes such as tissue repair and remodelling and may be implicated in pathological conditions, such as tumour vessel proliferation. Regulation of VEGF levels in pregnancy facilitates the rapid expansion¹² of placental vasculature to support the growing fetus. Fibroblast Growth Factor (FGF) is another such mediator. It promotes remodelling of the extracellular matrix, enabling endothelial cell migration to form new vessels. FGF also contributes significantly to placental vascularization, influencing the differentiation of trophoblast cells, which are essential for establishing and maintaining the placenta. Platelet-derived growth factor PDGF, predominantly known for its role in the recruitment and growth of smooth muscle cells and fibroblasts, also contributes to the angiogenic process. PDGF aids in the stabilization and maturation of newly formed capillaries by promoting structural integrity. Platelet-derived growth factor (PDGF) has a nuanced role in placental development. It is involved in the recruitment and proliferation of pericytes and smooth muscle cells, vital for the stability and maturation of chorionic blood vessels and maintaining the foetal-maternal placental barrier. Deregulation or dysfunction

of these factors may predispose to complications of placental insufficiency, including preeclampsia, intrauterine growth restriction (IUGR), and foetal distress.

Low-dose aspirin (acetylsalicylic acid) is a widely used anti-inflammatory and antiplatelet agent that inhibits the cyclooxygenase (COX) enzymes, particularly COX-1, to reduce the synthesis of thromboxane A₂, a vasoconstrictor and platelet aggregator. It also improves endothelial function and may support angiogenesis and vascular adaptation. It is known to have a broad spectrum of therapeutic applications, including the management of cardiovascular disease¹³. Recently, a protective role in uteroplacental circulation has garnered significant interest. In a landmark meta-analysis by the Cochrane Collaboration, comparing low-dose aspirin with placebo or no treatment in pregnant women at risk of preeclampsia, there was a 17% reduction of adverse outcomes in women treated with aspirin. This included not only preeclampsia but the risk of intrauterine growth restriction (IUGR) and preterm birth. Preeclampsia is characterized by impaired placental angiogenesis, possibly related to suboptimal vascular invasion of the decidua by the cytotrophoblast in early implantation. This occurs during the first trimester. Research indicates that the timing of treatment with LDA is critical for its effectiveness in preventing preeclampsia. The consensus recommends that it begins before 16 weeks, with some guidelines advocating as early as 12 weeks. This suggests that the angiogenic properties of LDA and its effect on tissue growth factors and inflammatory pathways account for its clinical benefit. Studies now also support the prophylactic use of LDA in women with a history of chronic hypertension, renal disease, autoimmune diseases, diabetes mellitus, and multiple gestations to improve pregnancy outcomes¹³. While there is little doubt that low-dose aspirin improves placental blood flow, there is, however, a lack of direct evidence to demonstrate a unique benefit in pregnancies affected specifically by SUA. Despite this, most guidelines recommend the routine use of LDA in this setting. As in all cases, however, the decision to use low-dose aspirin should be individualized, considering the overall risk profile rather than the presence of SUA alone.

Finally, while numerically, a single umbilical artery may physically limit the cross-sectional area available for blood flow and, thus, the potential volume of placental blood supply, the vessel is not necessarily analogous

to the dual arteries of a normal cord. It is wider, tending to have a larger diameter, providing a compensatory increase in expected blood flow. This is described by Poiseuill's Law, which shows that fluid flow will rise by a factor of 2 to the power of 4, or 16, for each unit of increase in a vessel's diameter.

Cord events

The mechanical strength of the umbilical cord, referring to its resistance to compression and stretching and its vulnerability to knotting or entanglement, depends primarily on the structural integrity of the Wharton's jelly and the collagenous sheath surrounding the vessels rather than the number of arteries. While SUA is a variation from the typical anatomical structure, it does not inherently confer a weaker or less resilient cord in terms of physical strength¹⁴. Similarly, as noted earlier, the cord vessels are coiled along their length with as many as 40 helical turns as they journey between the foetus and the placenta. These are said to improve blood flow, and whilst they may also contribute to cord stability, the overall structural and functional benefits are not significantly diminished in the setting of a SUA.

Management: Antenatal care

Pregnancies complicated by SUA require close clinical surveillance. First-trimester screening should be considered to evaluate the risk of aneuploidy. A detailed morphology scan, including a foetal echocardiogram, should be performed to exclude congenital abnormalities. Thereafter, serial ultrasound assessment of intra-uterine growth and foetal wellbeing should be an essential adjunct to close clinical monitoring of mother and baby. Ultrasound imaging allows evaluation of fetal development, amniotic fluid, and ongoing surveillance of foetal wellbeing, including blood flow through the umbilical vessels. Continuous Doppler ultrasound measures blood velocity and resistance to flow through the umbilical arteries. It is described in terms of the Systolic/Diastolic (S/D) ratio, Pulsatility Index (PI), and Resistance Index (RI). These, in turn, predict the amount of vascular resistance in the placenta. A high resistance indicates placental insufficiency and may be associated with intrauterine growth restriction. These measurements may differ in the setting of a single umbilical artery²; however, repeated examination will still reveal a pattern of either change or preservation of flow parameters that can be interpreted to determine status or change in placental function.

Intrapartum care

Decisions regarding birth and mode of delivery are collaborative, involving recognition of antenatal risk, previous obstetric history and maternal choice². If natural birth is desired, the presence of a single umbilical artery may harken a risk of intrapartum foetal distress and thus advocate close foetal monitoring.

Postpartum care

After the baby is born, a thorough neonatal examination should attend to any abnormalities disclosed or identified during antenatal screening. The examination should consider the possibility of missed pathology and endeavour to exclude conditions of risk associated with SUA, particularly cardiac and genito-urinary.

Conclusion

SUA is a common anomaly affecting as many as 1% of all pregnancies. It is often silent, a coincidental finding in an otherwise normal pregnancy for which no sequela arises. It may, however, be associated with risk. The change to placental and foetal circulation may be part of a broader condition reflecting an abnormality of chromosomal disjunction, leading to a risk of trisomy or congenital abnormalities of cardiac, genitourinary, and gastrointestinal development. Even as an isolated finding, a SUA may be associated with an incarceration of placental function, leading to an impairment of growth support and foetal wellbeing. These concerns highlight the importance of sustained, collaborative care. If problems are identified, ongoing communication with appropriate partnering specialities can best inform and prepare caregivers to optimize timing and place of birth.

The administration of low-dose aspirin in SUA pregnancies has enamoured best practice due to its benefits in mitigating adverse outcomes, including intrauterine growth restriction (IUGR), preterm birth, and preeclampsia, conditions that may arise in response to impaired placental function. In all things, however, compliance with medical care is pivotal to the efficacy of treatment protocols and may be compromised by patient apprehension and misunderstanding. Our case demonstrates how this can be accentuated in regional or remote locales, where socio-economic disadvantages, linguistic barriers, and the unique challenges faced by immigrants or newly arrived citizens

exacerbate the potential for non-compliance. In these settings, adopting strategies centred on collaboration, engagement, inclusiveness, and empowerment becomes not only advantageous but imperative. By cultivating a healthcare milieu that prioritizes these principles, practitioners can significantly mitigate the barriers to adherence. Such an approach clarifies medical directives and vests patients with a sense of agency in their healthcare journey. Through a bespoke, patient-centric methodology that acknowledges and integrates the heterogeneous backgrounds of patients, healthcare professionals can amplify adherence rates, ensuring equitable care delivery across diverse demographic and geographic spectra. It is not enough to rescind our responsibility by saying a patient has not attended; we must try to reach further, to embrace and resolve the barriers that stop them from doing so. If we can do this, we provide our greatest asset, the ability to sit face-to-face and communicate.

Ongoing, attentive antenatal care is critical for patients at risk of adverse outcomes. Recognition of maternal wellbeing as a milieu for normal fetal growth can be assessed by regular face-to-face consultation, history-taking, and clinical examination to detect signs of evolving morbidity, such as a rise in blood pressure, a diminution of foetal movements or static fundal height measurements may warn of impending harm. Safe, bedside monitoring by caregivers skilled in point-of-care USS can provide opportunistic evaluation of critical markers of intrauterine condition such as umbilical artery dopplers, AFI, and foetal growth parameters¹⁴. Competency in these skills should become a priority of clinical training in remote hospital settings where resources for ancillary services are often tenuous and far removed.

This case review reflects on a condition of pregnancy that may affect maternal and foetal outcomes. It identifies recommendations for best practice and acknowledges the challenges of instigating such care in settings where geographical isolation or demographic vulnerability may make it hard to do so. It advocates for tolerance and collaboration with others in a partnership of care that includes the patient⁹. Finally, it recommends training and competency in bedside USS techniques as an opportunistic and immediate determination of foetal status to guide decision-making when and where it matters.

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