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Overview of Ultrasound and Color Doppler Imaging in predicting of Placenta Accreta Spectrum

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Abstract

Placenta accreta spectrum (PAS) is one of the most dangerous conditions associated with pregnancy, because hemorrhage may result in multisystem organ failure, disseminated intravascular coagulation, need for admission to an intensive care unit, hysterectomy, and even death. The etiology of PAS remains controversial. Previous Cesarean section and placenta previa are the two most common risk factors. Early diagnosis of PAS aids in improvement of maternal outcomes through multidisciplinary planning. The screening process offers perfect opportunities for them and their families to be counseled about the suspected abnormal placentation. Current challenges include the accurate detection of PAS in the first trimester. The aim of this article study was to review the role of ultrasound and Color Doppler Imaging in predicting of PAS.

Keywords: Placenta Accreta Spectrum ; Ultrasound ; Color Doppler Imaging

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1. Introduction

Placenta accreta spectrum (PAS) is the general term applied to abnormal adherence of the placental trophoblast to the uterine myometrium; it is also referred to as morbidly adherent placenta. The spectrum includes placenta accreta (attachment of placenta to the myometrium without intervening decidua), placenta increta (invasion of the trophoblast into the myometrium), and placenta percreta (invasion through the myometrium, serosa, and into surrounding structure [1]. Rates of placenta accreta spectrum are increasing. Observational studies from the 1970s and 1980s described the prevalence of placenta accreta as between 1 in 2,510 and 1 in 4,017 compared with a rate of 1 in 533 from 1982 to 2002 [2]. In 2016, the National Inpatient Sample found that the overall rate of placenta accreta in the United States was 1 in 272 for women who had a birth-related hospital discharge diagnosis, which is higher than any other published study [3]. The increasing rate of placenta accreta over the past four decades is likely due to a change in risk factors, most notably the increased rate of cesarean delivery [4]. There are several risk factors for placenta accreta spectrum. The most common is a previous cesarean delivery, with the incidence of placenta accreta spectrum increasing with the number of prior cesarean deliveries [5]. The rate of placenta accreta spectrum increased from 0.3% in women with one previous cesarean delivery to 6.74% for women with five or more cesarean deliveries [6]. Additional risk factors include advanced maternal age, multiparity, prior uterine surgeries or curettage, and Asherman syndrome [7].

Placenta previa is another significant risk factor. Placenta accreta spectrum occurs in 3% of women diagnosed with placenta previa and no prior cesarean deliveries. In the setting of a placenta previa and one or more previous cesarean deliveries, the risk of placenta accreta spectrum is dramatically increased. For women with placenta previa, the risk of placenta accreta is 3%, 11%, 40%, 61%, and 67%, for the first, second, third, fourth, and fifth or more cesarean, respectively [8]. Moreover, abnormal results of placental biomarkers increase the risk of placenta accreta spectrum. For example, unexplained elevation in maternal serum alpha fetoprotein is associated with an increased risk of placenta accreta spectrum [9]. However, maternal serum alpha fetoprotein is a poor predictor of placenta accreta spectrum and is not accurate enough to be clinically useful. Other placental analytes linked to placenta accreta spectrum include pregnancy-associated plasma protein A, pro B-type natriuretic peptide, troponin, free β-hCG (mRNA), and human placental lactogen (cell-free mRNA) [10]. In addition, other proposed markers of aberrant trophoblast invasion, such as total placental cell-free mRNA, may be associated with placenta accreta spectrum. As with alpha fetoprotein, they are too nonspecific for clinical use [11]. The most favored hypothesis regarding the etiology of placenta accreta spectrum is that a defect of the endometrial–myometrial interface leads to a failure of normal decidualization in the

area of a uterine scar, which allows abnormally deep placental anchoring villi and trophoblast infiltration [12].

Several studies suggest that disruptions within the uterine cavity cause damage to the endometrial–myometrial interface, thereby affecting the development of scar tissue and increasing the likelihood of placenta accreta [13]. However, this explanation fails to explain the rare occurrence of placenta accreta spectrum in nulliparous women without any previous uterine surgery or instrumentation [1].

2. Clinical presentation and prenatal screening:

Usually, the first clinical presentation for PAS is massive obstetric hemorrhage occurring during delivery, when attempting to remove the placenta manually. Notably, antenatal bleeding may be observed among those women with placenta previa. Besides, presenting with abdominal pain is sometimes a warning of uterine rupture, probably as a consequence of placenta percreta [14]. For those women who are asymptomatic (most without certain risk factors), obstetric ultrasound examination may have some suspected findings. Routine prenatal screening is essential for early detection of PAS especially in women with less prominent risk factors [15]. Placenta accreta should be suspected in women who have both a placenta previa, particularly anterior, and a history of cesarean or other uterine surgery. The most important factor affecting outcome is prenatal diagnosis of this condition. It gives the opportunity to make a delivery plan that properly anticipates the expected blood loss and other potential complications of delivery [16]. In addition, it gives the opportunity for electively timing the procedure since prevention of complications ideally requires the presence of a multidisciplinary surgical team. Antenatal ultrasound is the technique of choice used to establish the diagnosis and guide clinical management [17]. Signs of accretion may be seen as early as in the first trimester**.** The ultrasound examinations performed up to 10 gestational weeks among women later proven to have placenta accreta on pathological examination. All had low-lying gestational sacs which are clearly attached to the uterine scar. The myometrium was thin in the area of the scar to which the sac was attached compared to normal early gestational sacs [18].

Second and third trimester gray-scale sonographic characteristics include loss of continuity of the uterine wall, multiple vascular lacunae (irregular vascular spaces) within placenta, giving “Swiss cheese” appearance adjacent to the placental implantation site, lack of a hypoechoic border (myometrial zone) between the placenta and the myometrium, bulging of the placental/myometrial site into the bladder, and increased vasculature evident on color Doppler sonography [19]. If the ultrasound findings are not considered definitive, or the placenta is located on the posterior wall, magnetic resonance imaging can be performed using gadolinium contrast intravenously. Magnetic resonance imaging findings considered suspicious for the presence of placenta accreta include placental heterogeneity, mass effect of the placenta into the underlying bladder or extending laterally or posteriorly beyond the normal uterine contour, obliteration of the myometrial zone visible on initial uptake of gadolinium, and a beading nodularity within the placenta [20]. Other than the imaging methods, elevated biochemical markers in maternal serum such as elevated levels of alpha fetoprotein and human chorionic gonadotropin within the triple screening test have been reported to be associated with an increased risk of placenta accreta. Though the mechanism is unclear, abnormality of the placental-uterine interface that may lead to leakage into the maternal circulation may explain this increase [21]. At this time no antenatal diagnostic technique affords the clinician 100% assurance of either ruling in or ruling out the presence of placenta accreta. The definitive diagnosis of placenta accreta is usually made postpartum on hysterectomy specimens when an area of accretion shows chorionic villi in direct contact with the myometrium and absence of decidua [22].

3. How to diagnose PAS?

Two-dimensional ultrasound evaluation, by transabdominal and transvaginal approaches, with gray scale and color Doppler imaging (CDI), is the recommended first-line modality for antenatal diagnosis of PAS [1].Gray-scale and CDI are considered the primary diagnostic tools, with overall sensitivity and specificity of 82.4%–100% and 71%–100%, respectively [23].

- **Gray- scale US features:**

(a) Abnormal placental lacunae:

Presence of multiple lacunae, which are large irregularly, shaped placental hypoechoic spaces that give the placenta a moth-eaten appearance (Fig.1); they can contain internal turbulent flow at gray-scale US Lacunae are usually centered within a lobule or cotyledon and are adjacent to the involved myometrium. These are not to be confused with placental lakes, which are features of a normal placenta and appear as a few small hypoechoic spaces with regular margins scattered throughout the placental tissue with slow flow [24].

(b) Loss of retroplacental clear zone:

Marked by obliteration or irregularity of the hypoechoic plane between the placenta and myometrium (Fig. 2). This area can be falsely obscured by pressure from a distended urinary bladder and excess pressure from the ultrasound transducer [25].

(c) Myometrial thinning:

Asymmetric thinning of myometrium overlying the placenta relative to myometrium not covered by placenta (Fig. 3). This can be focal or diffuse, with the myometrium measuring <1 mm or being nonvisible. It is associated with prior hysterotomy scars or placental invasion [26].

Placental bulge:

Focal bulge of the placenta in an area of myometrial thinning (Fig. 3), leading to deviation of the uterine serosa from its expected plane and uterine contour deformity [27].

(d) Bladder wall interruption:

Disruption of the normally smoothly echogenic bladder wall, located between the uterine serosa and urinary bladder lumen [28].

(f) Focal exophytic mass:

Focal extension of placental trophoblastic tissue beyond the uterine serosa (Figure 4), most commonly into the bladder [29].

- ***Color doppler us features:***

i- bridging vessels:

Perpendicularly oriented vessels seen extending from the placenta through the underlying myometrium and Into the uterine serosa or extrauterine structures, including but not limited to the bladder [30].

ii-Uterovesical hypervascularity:

Increased color Doppler signal between the myometrium and posterior bladder wall (Figure 5), which likely indicates increased vascular density and tortuosity in that region [31].

iii-Subplacental hypervascularity:

Increased color Doppler signal in the placental bed, which likely indicates increased vascular density and tortuosity in that region [32].

In more recent studies; showed fetal arteries that traverse the placental width have been described and been correlated to PAS (Figure 6), the transplacental fetal vessel is strongly correlated with PAS and suggest that this corresponds either to an immature fetal villous or a ruptured anchoring villous, both of which have been described in pathologic reports of PAS [33].

4. Clinical Significance:

An accurate and an early prenatal diagnosis of PAS allow time for a multidisciplinary team to plan delivery in a center with expertise in surgical management of these disorders. As such, surgical complications, blood loss, requirement for transfusion, hysterectomy, and intensive care unit stay can be reduced in women after antenatal diagnosis, compared with those in whom the diagnosis of PAS is made during the intrapartum period. Ultrasound (US) prediction of placenta accreta spectrum (PAS) in the first trimester may be aided by postprocessing mechanisms employing color pixel quantification near the bladder–uterine serosal interface. Color Doppler mapping in the first trimester showed an increase in color pixel area near the bladder–uterine serosal interface in women requiring cesarean hysterectomy for PAS, compared to women without hysterectomy or pathologic invasion. This finding of low velocity at the junction of the uterine scar with the trophoblast implantation has the potential to be further automated and quantified to become a strong predictor of future complications in pregnancy.

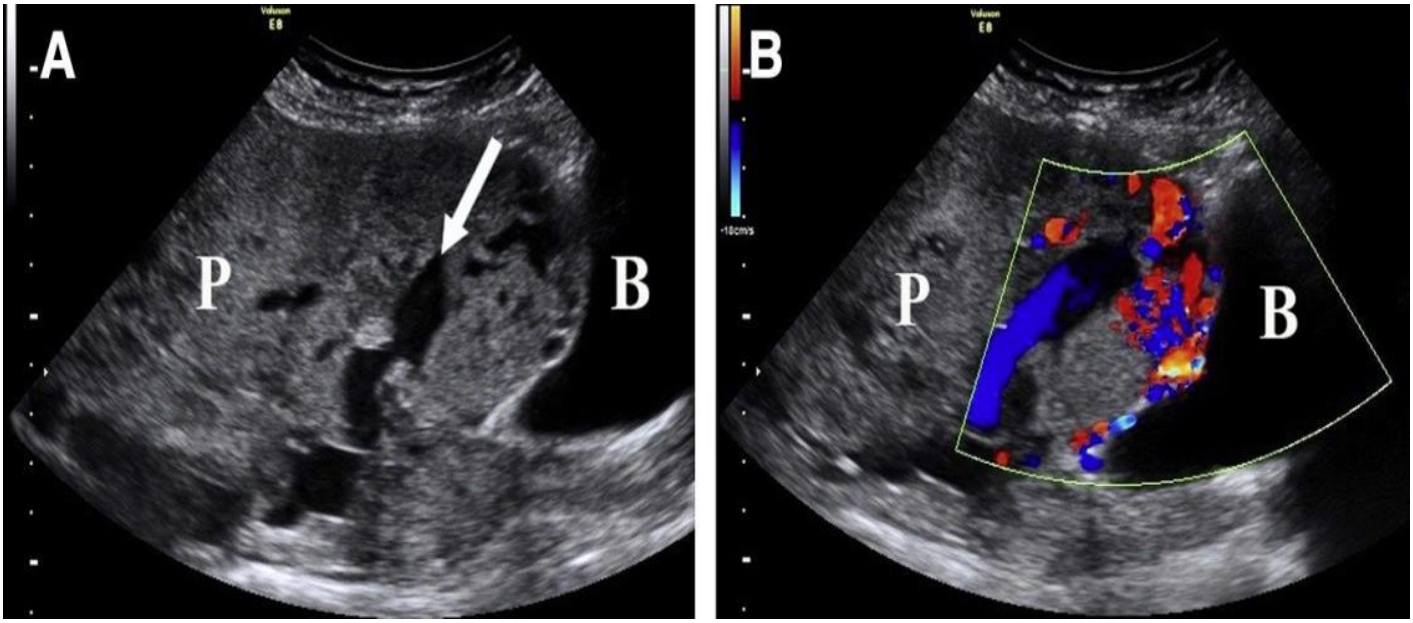

Figure 1: Transabdominal ultrasound longitudinal views of placenta (P) previa accreta at 36 weeks. A, “Moth- eaten” area with numerous lacunae of different size and shape secondary; and B, high-velocity, turbulent blood flow within lacunae on color Doppler imaging next to bladder (B) [24]

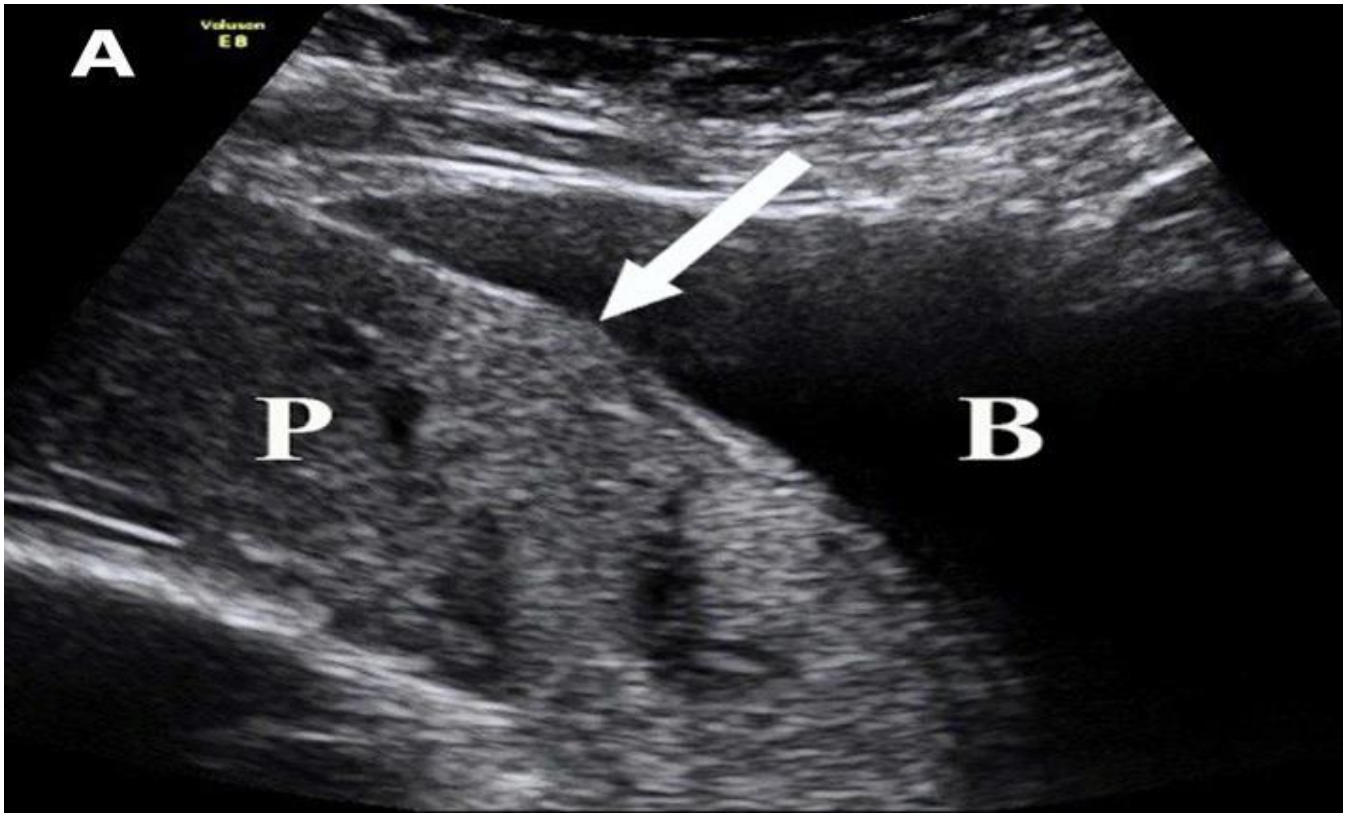

Figure 2: Myometrial thinning secondary to uterine thinning at scar defect. A, Transabdominal ultrasound longitudinal view of placenta (P) previa at 36 weeks showing myometrium defect (arrow) under bladder (B). Note absence of clear zone and myometrium in area (B**)** [26].

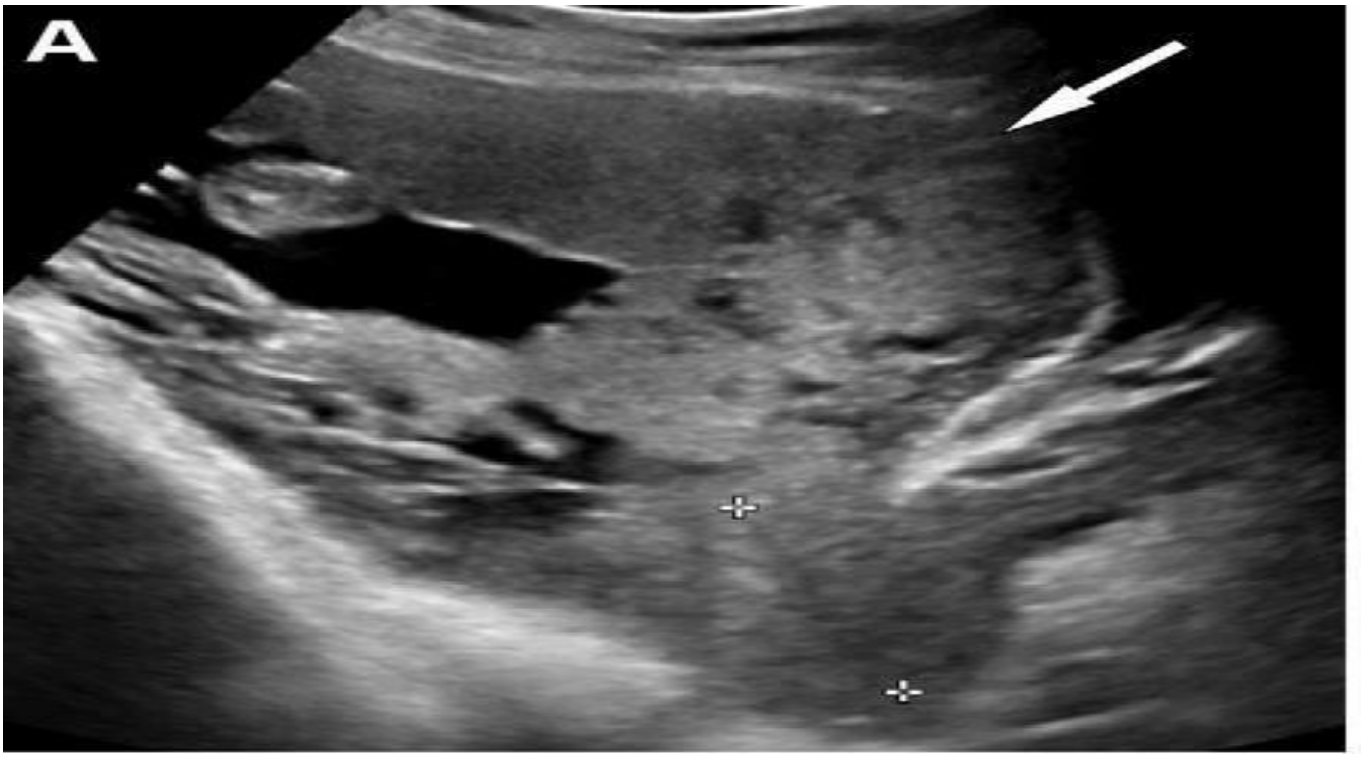

Figure 3: Myoinvasive PAS disorder in a 31-year-old patient who presented at 32 weeks gestation with vaginal bleeding. transabdominal US images of the placenta show thinned myometrium over the anterior placenta and placental bulge [24].

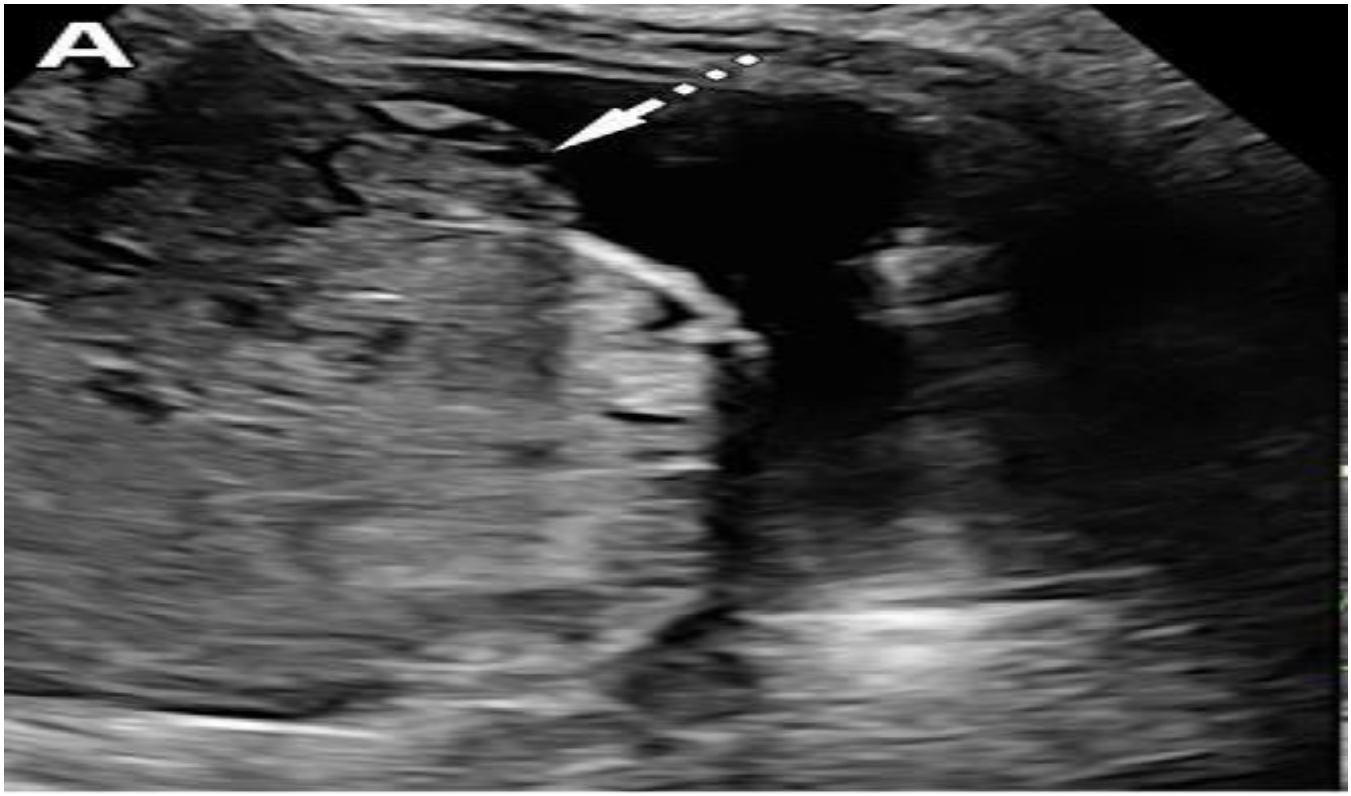

Figure 4: Myoinvasive PAS disorder in a 32-year-old patient with a history of three prior cesarean sections transabdominal US images of the uterovesical junction show an exophytic mass (dashed arrow) [24]

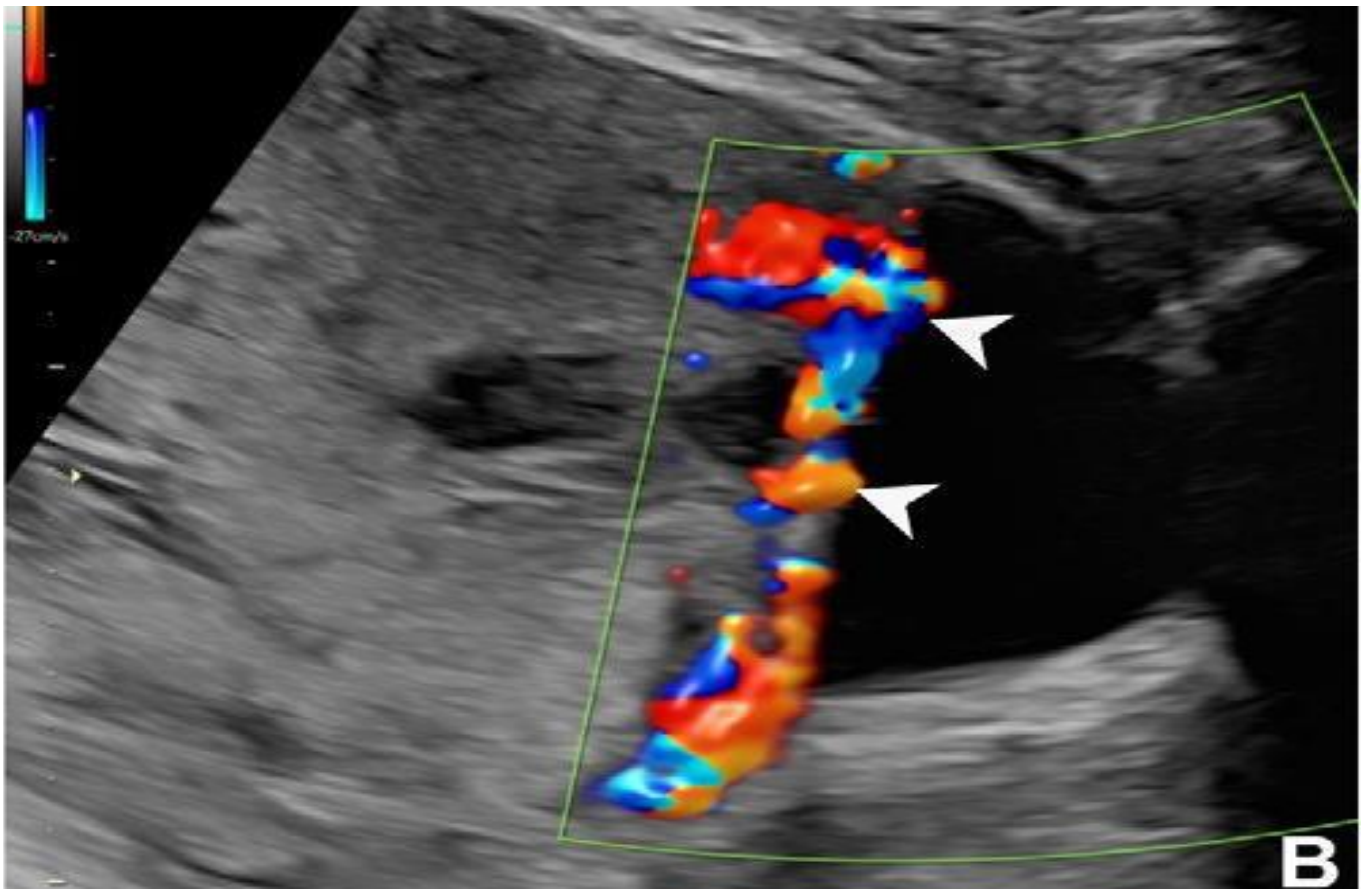

Figure 5: Uterovesical hypervascularity [24]

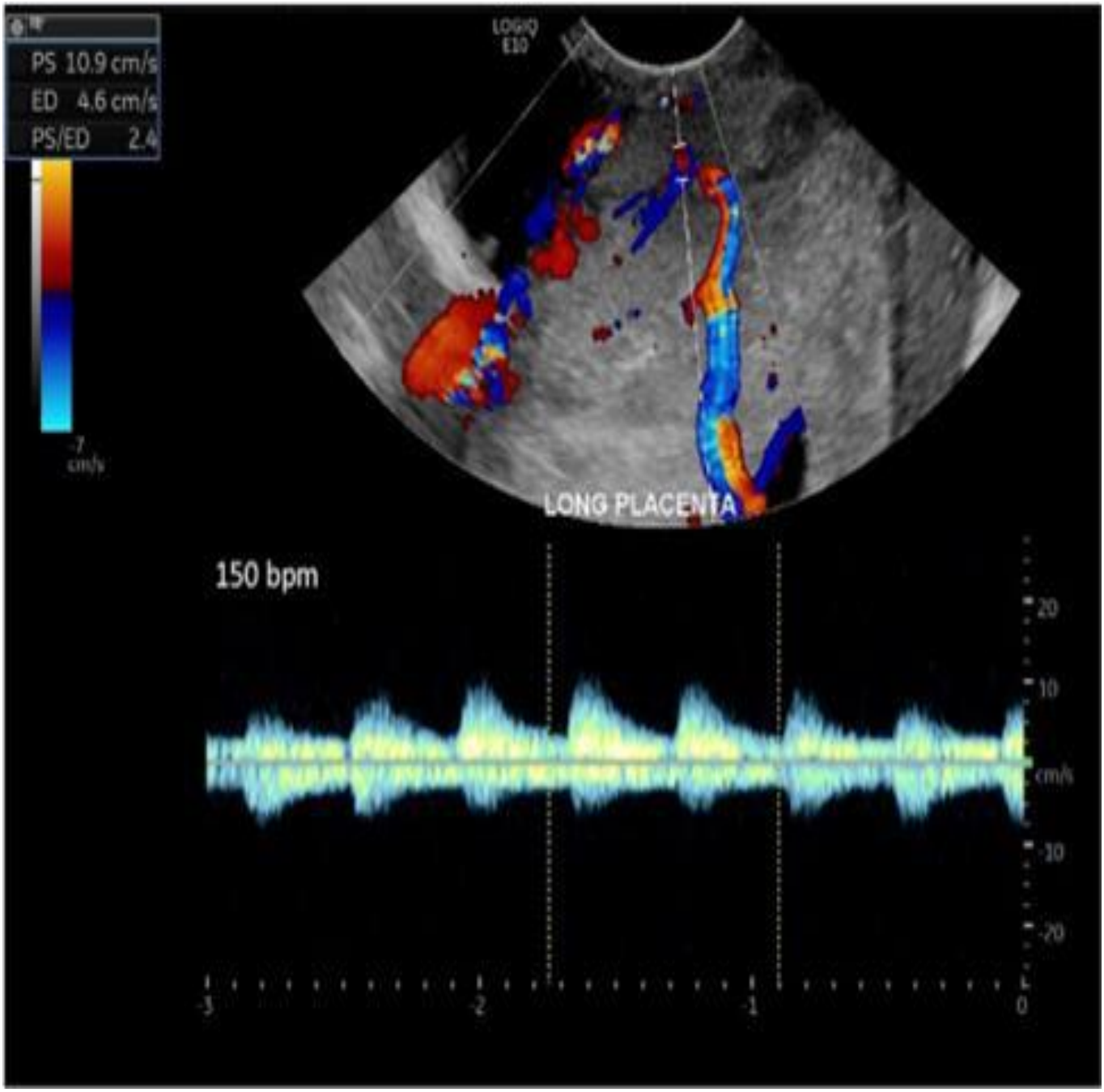

Figure 6: A transplacental fetal artery in a patient with placenta FIGO Grade 2 (increta). Color and spectral Doppler image shows a fetal artery (150 bpm) traversing the width of the placenta nearly to the basal plate Ooted from [33].

5. Conclusions

Placenta accreta spectrum (PAS) refers to an abnormally invasive implantation of the placenta into the uterine myometrium. Prenatal ultrasound, including color Doppler examination, is a promising diagnostic tool for PAS. Prenatal care and timely diagnosis of PAS would significantly improve eventual outcomes, such as less blood loss and fewer transfusions of blood products, compared with those diagnosed at delivery.

Conflict of interest

The authors declare no conflict of interest.

Author contribution

Authors contributed equally in the study.

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