International Journal of Clinical Obstetrics and Gynaecology

ISSN (P): 2522-6614
ISSN (E): 2522-6622
Indexing: Embase
Impact Factor (RJIF): 6.71

www.gynaecologyjournal.com
2026; 10(1): 101-112
Received: 09-11-2025
Accepted: 13-12-2025

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Caesarean scar ectopic pregnancy: Pathogenesis, diagnosis and management strategies

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DOI: https://www.doi.org/10.33545/gynae.2026.v10.i1b.1859

Abstract
Caesarean scar ectopic pregnancy (CSP) represents a rare but potentially catastrophic complication of modern obstetrics, characterised by blastocyst implantation within the fibrotic tissue of a previous hysterotomy scar. Mirroring the global rise in caesarean deliveries, CSP incidence is escalating, posing severe risks of uterine rupture and life-threatening haemorrhage. This comprehensive review elucidates the pathogenesis of CSP, identifying mechanisms such as microscopic scar dehiscence, "uterine niches," and aberrant trophoblastic invasion into poorly vascularized tissue. We critically evaluate diagnostic challenges, affirming transvaginal ultrasonography (TVS) as the primary imaging modality, while highlighting the utility of MRI for superior anatomical delineation and preoperative planning. Therapeutic strategies are systematically examined, contrasting conservative medical management using methotrexate both systemic and local with definitive surgical interventions, including laparoscopic and hysteroscopic resection. Furthermore, the integration of adjunctive technologies like uterine artery embolization (UAE) is discussed as a vital measure for haemorrhage control and fertility preservation. The review concludes by addressing the long-term reproductive implications of CSP, including the risk of placenta accrete spectrum disorders, and advocates for a multidisciplinary approach to optimize maternal safety and improve clinical outcomes.

Keywords: Caesarean scar pregnancy (CSP), ectopic pregnancy, transvaginal ultrasonography, uterine rupture, methotrexate, uterine artery embolization (UAE), hysteroscopy

Caesarean scar ectopic pregnancy (CSP) represents an uncommon yet clinically significant form of ectopic gestation, wherein the blastocyst implants within the fibrotic tissue of a previous caesarean section scar [1]. First documented in the 1970s, CSP has garnered increasing attention in recent decades, primarily due to the rising global incidence of caesarean deliveries and a parallel escalation in reported cases [2]. Although it constitutes only a small fraction of all ectopic pregnancies, the potential for catastrophic maternal morbidity underscores its importance as a distinct clinical entity in modern obstetrics [3].

The estimated incidence of CSP ranges from 1 in 1,800 to 1 in 2,214 pregnancies, significantly lower than that of tubal ectopic pregnancies, which can occur in approximately 1 in 100 pregnancies in certain populations [4]. Despite its relative rarity, the potential for rapid deterioration due to uterine rupture or severe haemorrhage highlights the necessity of prompt recognition and management [5]. The upward trend in CSP incidence closely mirrors the global increase in caesarean section rates, establishing a clear epidemiological link between the two phenomena [6].

A mechanistic understanding of CSP pathogenesis is essential for improving diagnostic and therapeutic outcomes [7]. The defining feature is the abnormal implantation of the blastocyst into a microscopic defect within the myometrium or fibrous tissue at the site of a prior hysterotomy scar [8]. Several mechanisms have been postulated, including micro tubular tracts within scar dehiscence or aberrant implantation onto scarred endometrial tissue, possibly secondary to fibrosis or localised ischemia [9]. Such aberrant trophoblastic invasion into poorly vascularized tissue predisposes to inadequate placentation, uterine rupture, and life-threatening haemorrhage [10].

Identification of predisposing factors is vital for early detection and risk stratification. Previous caesarean deliveries remain the strongest independent risk factor, particularly with multiple prior procedures [11]. The risk increases exponentially with each successive caesarean section, with women having two or more previous caesarean deliveries demonstrating a significantly elevated

incidence of CSP compared to those with a single prior procedure [12]. Other contributory factors include previous uterine surgeries such as myomectomy or cornual resection, advanced maternal age, and the use of assisted reproductive technologies (ART) [13].

The clinical diagnosis of CSP is often challenging due to its nonspecific presentation, which can mimic a normal intrauterine pregnancy or an abortion in progress [14]. Common symptoms, including vaginal bleeding and lower abdominal pain, are insufficiently distinctive to confirm diagnosis [15]. The absence of pathognomonic clinical features necessitates a high index of suspicion, particularly in women with a history of caesarean delivery presenting with early pregnancy complications [16].

Transvaginal ultrasonography (TVS) is the cornerstone diagnostic modality, providing high-resolution visualisation of a gestational sac embedded within the anterior lower uterine segment at the site of the caesarean scar [17]. Sonographic features typically include an empty uterine cavity, a gestational sac located within the anterior uterine isthmus, and a thin or absent myometrial layer between the sac and the bladder, often visualised on sagittal imaging [18]. When ultra-sonographic findings are inconclusive, magnetic resonance imaging (MRI) offers superior delineation of myometrial integrity and spatial relationships, facilitating both diagnosis and preoperative planning [19].

The clinical significance of CSP extends beyond immediate maternal morbidity to encompass long-term reproductive implications [20]. Women who have experienced CSP face increased risks in subsequent pregnancies, including recurrent CSP, placenta accrete spectrum disorders, and uterine rupture [21]. These potential complications underscore the importance of comprehensive counselling regarding future fertility and pregnancy planning following CSP diagnosis and treatment [22].

This comprehensive review seeks to investigate the underlying mechanisms, diagnostic challenges, and therapeutic approaches to managing CSP. Additionally, it will showcase the most recent advancements in diagnostic tools and treatment procedures, and explore future research pathways required to enhance patient outcomes [23].

2. Pathogenesis and Contributing Factors

2.1 Etiological Theories of CSP

Caesarean scar ectopic pregnancy (CSP) arises when a fertilised ovum implants within the fibrotic scar tissue of a prior caesarean incision, rather than in the healthy endometrial cavity. This aberrant implantation is of particular concern because the scar's deficient vascularisation and compromised mechanical integrity predispose to catastrophic outcomes such as uterine rupture and severe haemorrhage [24]. A precise understanding of CSP pathogenesis is therefore essential for improving diagnostic accuracy, refining therapeutic strategies, and mitigating maternal morbidity [25].

One prevailing theory proposes that CSP develops when the blastocyst gains access to the myometrium through a microscopic dehiscent tract within the previous hysterotomy scar. Such tracts may result from suboptimal surgical closure, delayed epithelialisation, or incomplete tissue healing following a caesarean section [26]. These microdefects act as potential sites of implantation, enabling trophoblastic invasion into the fibrotic scar [27].

The quality of uterine scar healing is influenced by multiple factors, including surgical technique, suture material, degree of haemostasis achieved during closure, and individual patient healing characteristics [28]. Single-layer versus double-layer closure techniques have been debated in the literature, with some studies suggesting that single-layer closure may be associated with an increased risk of scar defect formation [29]. Additionally, the presence of infection, haematoma formation, or tissue devitalisation at the time of caesarean delivery can compromise scar integrity and increase susceptibility to abnormal implantation [30].

An alternative model suggests that implantation occurs when the blastocyst attaches to a fibrotic scar surface rather than a properly decidualised endometrial lining [31]. Because scar tissue lacks the cyclical morphological and vascular adaptations of normal endometrium, trophoblastic invasion occurs in an environment characterised by inadequate perfusion and poor structural support [32]. This aberrant milieu facilitates partial trophoblastic penetration into the myometrium and may lead to early uterine rupture or uncontrolled haemorrhage [33].

The molecular and cellular mechanisms underlying abnormal implantation in scar tissue are increasingly being elucidated [34]. Studies have demonstrated altered expression of adhesion molecules, growth factors, and angiogenic factors in caesarean scar tissue compared to normal endometrium [35]. These molecular alterations may create a permissive environment for trophoblastic invasion while simultaneously impairing the normal decidualisation response that would otherwise limit invasion depth [36].

Emerging evidence also implicates "uterine niches" small fluid-filled indentations or defects in the lower uterine segment scar as microenvironments that favour abnormal implantation [37]. These niches can collect menstrual blood, cellular debris, or inflammatory exudate, creating a biochemical milieu that enhances blastocyst adherence and implantation [38]. Hysteroscopic and sonographic studies have demonstrated that uterine niches are present in a substantial proportion of women following caesarean delivery, though not all women with niches develop CSP [39].

The "wedge theory" represents another mechanistic explanation, proposing that the blastocyst becomes wedged into a microscopic tract or defect during its passage through the endocervical canal and lower uterine segment [40]. This theory is supported by the observation that CSP most commonly occurs at the site of the hysterotomy incision, typically in the lower anterior uterine segment [41].

2.2 Risk Factors Predisposing to CSP

Comprehensive identification of CSP risk factors is critical for early diagnosis, risk stratification, and prevention [42]. Understanding these factors not only clarifies disease pathophysiology but also informs safer obstetric and surgical practices [43].

A history of multiple caesarean deliveries represents the most significant and consistent risk factor. The cumulative formation of fibrotic scars, combined with potential suboptimal healing or technical variation in closure, exponentially increases the likelihood of implantation within a myometrial defect [44]. Each successive caesarean section augments the probability of microstructural discontinuities within the uterine wall, predisposing to abnormal nidation [45].

The interval between the most recent caesarean delivery and the index pregnancy also appears to influence CSP risk [46]. Shorter interpregnancy intervals may not allow sufficient time for complete scar maturation and remodelling, potentially leaving residual defects that facilitate abnormal implantation [47].

Other uterine surgical interventions, including myomectomy

(fibroid excision) and cornual resection (removal of uterine horns), have also been implicated [48]. These procedures involve incisions that may heal imperfectly, leaving areas of tissue weakness vulnerable to implantation [49]. Dilatation and curettage procedures, particularly when performed for management of retained products of conception or termination of pregnancy, may cause endometrial trauma that predisposes to subsequent ectopic implantation [50].

The use of assisted reproductive technologies (ART), particularly *in vitro* fertilisation (IVF), is another important risk factor [51]. ART procedures often entail uterine instrumentation and manipulation, which may alter endometrial receptivity or cause microscopic trauma to the uterine wall [52]. Additionally, embryo transfer performed in proximity to a caesarean scar increases the probability of aberrant implantation [53].

Advanced maternal age has been identified as an independent risk factor for CSP, possibly related to age-related changes in endometrial receptivity and uterine vascularity [54]. Women over 35 years of age demonstrate a higher incidence of CSP compared to younger women, even after controlling for parity and number of previous caesarean deliveries [55].

Uterine structural abnormalities whether congenital, such as a bicornuate uterus or septate uterus, or acquired, such as those associated with endometriosis or chronic infection also modify the uterine architecture, leading to abnormal implantation trajectories [56]. These pathologies can distort the uterine contour, interfere with normal trophoblastic migration, and increase susceptibility to implantation in scarred areas [57].

3. Diagnostic Challenges and Imaging Modalities

3.1 Clinical Presentation and Diagnostic Difficulties

Caesarean scar ectopic pregnancy (CSP) presents significant diagnostic challenges because of its infrequency and lack of pathognomonic symptoms, which frequently coincide with other complications in early pregnancy [58]. Women diagnosed with CSP commonly present with symptoms such as abdominal discomfort and vaginal bleeding, which are characteristic of many conditions, including tubal ectopic pregnancies, early pregnancy loss, and even normal intrauterine pregnancies [59].

The timing of symptom onset is variable, with some women remaining asymptomatic until relatively advanced gestational ages, while others present with acute symptoms in the early first trimester [2]. Asymptomatic cases may be detected incidentally during routine first-trimester ultrasonography, highlighting the importance of careful sonographic evaluation of the lower uterine segment in all women with prior caesarean delivery (Table 1) [2].

Physical examination findings are typically nonspecific and may include a slightly enlarged uterus, cervical motion tenderness [1]. In cases with significant haemorrhage, signs of hypovolemic shock, including tachycardia, hypotension, and pallor, may be present [5]. However, the majority of patients are hemodynamically stable at initial presentation, emphasising the importance of imaging studies for accurate diagnosis [2].

3.2 Ultra-sonographic Diagnosis

Transvaginal ultrasonography (TVS) is considered the primary method for diagnosing CSP because it can produce highly detailed images of the pelvic anatomy with excellent resolution [2]. TVS is essential for detecting critical diagnostic criteria, such as the precise location of the gestational sac within the lower uterine segment at the site of the previous caesarean scar [2].

The primary sonographic findings include an empty uterine cavity and cervical canal, a gestational sac located anteriorly at the level of the internal OS or isthmus, and absent or diminished myometrial tissue between the gestational sac and the bladder wall [2]. Additional supportive findings include increased vascularity around the gestational sac on colour Doppler imaging, often described as a "ring of fire" appearance due to prominent trophoblastic blood flow [16].

Measurement of the myometrial thickness between the gestational sac and the bladder is a critical diagnostic parameter [2]. A myometrial thickness of less than 2 mm is highly suggestive of CSP and indicates increased risk of uterine rupture and haemorrhage [5]. Some authors have proposed classification systems based on the degree of myometrial invasion, distinguishing between endogenic type (gestational sac growing toward the uterine cavity) and exogenic type (gestational sac growing toward the bladder and abdominal cavity), with the latter carrying a significantly worse prognosis [13].

The use of three-dimensional (3D) ultrasonography has improved visualisation of spatial relationships between the gestational sac and the scar tissue, enabling a clearer understanding of the implantation site [13]. Three-dimensional reconstruction allows for detailed assessment of the gestational sac volume, its relationship to surrounding structures, and the integrity of the myometrial layer [3].

Power Doppler ultrasonography provides additional valuable information regarding the vascularity of the implantation site [2]. Increased blood flow surrounding the gestational sac is characteristic of CSP and helps differentiate it from other conditions, such as cervical pregnancy or incomplete abortion [3].

3.3 Magnetic Resonance Imaging

When ultrasound findings are unclear or when more detailed anatomical information is required, clinicians turn to magnetic resonance imaging (MRI), a powerful diagnostic modality [19]. MRI is particularly effective in differentiating soft tissues and provides a comprehensive assessment of the extent of trophoblastic invasion and the structural integrity of the caesarean scar [20].

MRI offers superior contrast resolution compared to ultrasonography and is not limited by patient body habitus, bowel gas, or operator dependence [19]. T2-weighted sequences are particularly useful for delineating the gestational sac, myometrial integrity, and the relationship between the pregnancy and the bladder [20].

Specific MRI findings suggestive of CSP include a gestational sac located anteriorly in the lower uterine segment, loss of myometrial continuity at the site of the previous caesarean scar, and thinning or absence of the myometrial layer between the gestational sac and the bladder [19]. MRI is particularly valuable in cases where surgical intervention is planned, as it provides detailed anatomical information that guides surgical approach and helps anticipate potential complications [20].

3.4 Biochemical Markers

Serum beta-human chorionic gonadotropin (β-hCG) levels are routinely measured in the evaluation of early pregnancy complications, though they have limited diagnostic specificity for CSP [3]. The pattern of β-hCG rise may provide some diagnostic clues, with CSP typically demonstrating an appropriate rise for gestational age, in contrast to failing intrauterine pregnancies or some ectopic pregnancies where suboptimal rises are observed [5].

Serial β-hCG measurements are valuable for monitoring

treatment response and detecting persistent trophoblastic tissue following intervention [3]. After successful treatment, β-hCG levels should decline according to a predictable pattern, with failure to decline appropriately suggesting incomplete treatment or persistent trophoblastic disease [5].

4. Management Strategies

4.1 Expectant Management

Expectant management is reserved for carefully selected patients in whom the gestational sac is small, fetal cardiac activity is absent, and serum β-hCG concentrations are declining [23]. Such conservative observation requires rigorous criteria: the patient must be asymptomatic, hemodynamically stable, and fully compliant with close surveillance protocols [60]. Despite its theoretical appeal in preserving uterine integrity, this approach carries a substantial risk of sudden uterine rupture and massive haemorrhage; therefore, continuous monitoring through serial β-hCG assays and transvaginal ultrasonography is indispensable [61].

The rationale for expectant management is based on the natural history of some CSPs, which may undergo spontaneous regression without intervention [23]. However, this approach is controversial and is generally considered appropriate only in highly selected cases with very early diagnosis, small gestational sac size, absent cardiac activity, and declining β-hCG levels [23] (Table 2).

Patients managed expectantly require frequent follow-up visits with serial β-hCG measurements, typically every 48-72 hours initially, and weekly transvaginal ultrasonography to monitor gestational sac size and detect signs of rupture or haemorrhage [23]. The success rate of expectant management varies widely in the literature, ranging from 30% to 70% depending on patient selection criteria and definition of success [23].

4.2 Medical Management

Medical therapy represents the mainstay of conservative management, typically employing methotrexate (MTX) a folate antagonist that inhibits trophoblastic proliferation by blocking DNA synthesis [62]. MTX can be administered either by intramuscular injection route or locally via ultrasound-guided intra-sac injection [22].

Systemic methotrexate is typically administered using protocols adapted from those developed for tubal ectopic pregnancy, most commonly either a single-dose regimen (50 mg/m² body surface area) or a multi-dose regimen [22]. The single-dose protocol is simpler and associated with fewer side effects, while the multi-dose regimen may be more effective for larger gestational masses or higher β-hCG levels [22].

Local therapy offers targeted cytotoxic action with reduced systemic toxicity and is often supplemented with potassium chloride (KCl) or hyperosmolar glucose, which induce embryocidal and osmotic effects, respectively, to enhance overall efficacy [22]. Local injection of methotrexate is typically performed under ultrasound or laparoscopic guidance, with the medication injected directly into the gestational sac [22].

Patient selection remains crucial for medical management success, as MTX effectiveness is inversely correlated with gestational age, sac size, and initial β-hCG levels [62]. Generally, methotrexate is most successful when β-hCG levels are below 5,000 mIU/mL, gestational sac diameter is less than 4 cm, and cardiac activity is absent [62].

Monitoring during and after methotrexate treatment requires serial β-hCG measurements and ultrasonography [62]. Complications of methotrexate therapy include bone marrow suppression, hepatotoxicity, stomatitis, and gastrointestinal disturbances [62].

4.3 Surgical Interventions

Surgery remains the definitive approach for medical treatment failure, active bleeding, hemodynamic instability, or advanced gestational age [62]. The choice of surgical modality depends on the depth of myometrial invasion, gestational size, presence of cardiac activity, patient hemodynamic status, and the patient's reproductive preferences [62].

Laparoscopic resection is increasingly favoured as a minimally invasive approach, allowing precise excision of the gestational sac and concurrent repair of the uterine scar with reduced postoperative morbidity and faster recovery [63]. The laparoscopic approach provides excellent visualization of the pelvis and allows for meticulous dissection and haemostasis [62].

The advantages of laparoscopy include reduced postoperative pain, a shorter hospital stay, faster recovery, and improved cosmetic outcomes (Figure 1) compared to laparotomy [62]. However, laparoscopy requires advanced surgical skills and may not be appropriate in cases of hemodynamic instability, massive haemorrhage, or very large gestational masses [62].

Conversely, laparotomy is indicated in advanced cases involving large gestational sacs, extensive myometrial infiltration, hemodynamic instability, or suspected uterine rupture [62]. Laparotomy provides direct access and visualization of the uterus and allows for rapid control of haemorrhage [62].

Hysteroscopic resection offers another minimally invasive option, enabling direct visualisation and removal of residual trophoblastic tissue within the scar niche [64]. Hysteroscopy is particularly useful for endogenic-type CSPs where the gestational sac is growing toward the uterine cavity rather than toward the bladder [16]. When combined with preoperative methotrexate administration, hysteroscopy provides high efficacy with minimal blood loss and a lower risk of recurrence [64].

The primary risk of hysteroscopic management is uterine perforation, particularly when the myometrial layer between the gestational sac and bladder is very thin [64]. For this reason, hysteroscopy is generally reserved for cases with adequate myometrial thickness and endogenic-type implantation [64].

4.4 Adjunctive and Combined Approaches

Emerging interventional techniques have transformed the therapeutic landscape of CSP. Uterine artery embolization (UAE) is now widely utilized to devascularize the implantation site, thereby minimizing haemorrhage risk and facilitating safer subsequent surgical or medical treatment [65]. UAE involves selective catheterization of the uterine arteries under fluoroscopic guidance and injection of embolic material to occlude blood flow to the gestational sac [66].

UAE can be used as a primary treatment modality or as an adjunct to medical or surgical management [62]. The success rate of UAE varies depending on whether it is used alone or in combination with other treatments, with combination approaches generally demonstrating higher success rates [62]. Complications of UAE include post-embolization syndrome (pain, fever, nausea), infection, and rarely, uterine necrosis or infertility [62].

In refractory cases, adjunctive innovations such as balloon catheter tamponade offer effective control of post-excision bleeding by applying localized pressure to the uterine defect [67]. Moreover, combined laparoscopic-hysteroscopic approaches have demonstrated superior outcomes by enabling simultaneous

excision of the gestational sac, repair of the uterine wall, and detachment of the bladder from the lower uterine segment, thereby reducing operative injury and adhesion formation [68].
Ultimately, optimal management requires a multidisciplinary approach involving obstetricians, interventional radiologists, reproductive specialists, and anaesthesiologists to tailor treatment to the clinical condition of the patient and fertility goals [69].

5. Outcomes and Prognostic Implications

5.1 Maternal Morbidity and Mortality

CSP poses profound maternal risks due to the high vascularity and fragility of the implantation site, often leading to catastrophic haemorrhage that may necessitate emergency hysterectomy [70]. The risk of severe haemorrhage is particularly high in cases with deep myometrial invasion, large gestational sacs, or delayed diagnosis [71].
Uterine rupture, especially in advanced gestations, remains a critical life-threatening event with high transfusion and surgical morbidity rates [72]. The reported rate of hysterectomy for CSP ranges from 5% to 20% depending on the timing of diagnosis, treatment approach, and patient population [73].
Additional operative complications such as infection, pelvic adhesions, bladder injury, and need for blood transfusion further compound morbidity [74]. Bladder injury is a particular risk during surgical management due to the proximity of the implantation site to the bladder and the frequent presence of dense adhesions between the bladder and lower uterine segment [75].
Even under medical management, incomplete resolution or persistent trophoblastic tissue may precipitate delayed haemorrhage and require secondary surgical intervention [76].
Maternal mortality from CSP, while rare in settings with access to modern medical care, has been reported and typically results from uncontrolled haemorrhage or delayed diagnosis [77].

5.2 Impact on Fertility and Reproductive Outcomes

Preservation of future fertility is a central concern in CSP management. While methotrexate-based protocols aim to preserve uterine function, surgical excision though curative may compromise fertility potential by altering uterine architecture [78]. Postsurgical adhesions, particularly intrauterine adhesions (Asherman syndrome), can lead to menstrual abnormalities, infertility, and recurrent pregnancy loss [79].
Subsequent pregnancies carry increased risk of recurrent CSP, uterine rupture, and placenta accreta spectrum disorders due to residual scar weakness [18]. Women who have experienced CSP should be counselled regarding these risks and should undergo early ultrasonography in any subsequent pregnancy to confirm intrauterine implantation [23].
Placenta accreta spectrum disorders, including placenta accreta, increta, and percreta, represent particularly serious complications in pregnancies following CSP [80]. Despite these concerns, successful pregnancies following CSP treatment have been widely reported, with live birth rates ranging from 60% to 85% in women attempting conception after treatment [18].

5.3 Long-term Monitoring and Psychosocial Support

Long-term follow-up is imperative given that recurrence rates of CSP are estimated between 5% and 15% in subsequent pregnancies [62]. Post-treatment surveillance should include serial β-hCG measurements until normalization, typically defined as levels below 5 mIU/mL [3]. Early first-trimester transvaginal ultrasonography in subsequent pregnancies is essential to confirm intrauterine implantation and exclude recurrent CSP [18].
Beyond physiological recovery, psychological support is critical. The traumatic nature of CSP, potential fertility loss, and fear of recurrence frequently cause significant emotional distress [15]. Women who have experienced CSP may experience anxiety, depression, post-traumatic stress symptoms, and grief related to pregnancy loss or loss of fertility [15].
Comprehensive care therefore necessitates integrating psychological counselling and reproductive planning into post-treatment follow-up [4]. Preconception counselling is an important component of long-term care for women desiring future pregnancy [6].

6. Case Studies and Clinical Illustrations

6.1 Case Study 1: Combined Methotrexate and Hysteroscopy

A 34-year-old woman with four previous caesarean deliveries presented with minor lower abdominal discomfort and positive β-hCG levels eight months after her most recent caesarean section [81]. Transvaginal ultrasound revealed a gestational sac implanted in the anterior lower uterine segment at the site of the caesarean scar [2]. The management strategy involved initial systemic methotrexate administration followed by hysteroscopic suction evacuation five days later [82]. The procedure was successful with minimal haemorrhage and preservation of uterine architecture [83].

6.2 Case Study 2: Local Methotrexate with Potassium Chloride

A 29-year-old woman with two previous caesarean deliveries presented at six weeks gestation with vaginal bleeding and abdominal discomfort [2]. Transvaginal ultrasonography revealed a gestational sac with visible cardiac activity located in the lower anterior uterine segment at the caesarean scar site [2]. Given the patient's hemodynamic stability and desire for fertility preservation, a combined local treatment approach was selected, involving potassium chloride and methotrexate injection under ultrasound guidance [84]. Cardiac activity ceased immediately, and serial β-hCG monitoring demonstrated appropriate decline [65].

7. Future Directions and Research Needs

7.1 Advanced Diagnostic Technologies

The diagnosis of CSP has significantly improved with high-resolution imaging techniques, yet opportunities remain for earlier detection and more precise diagnosis [85]. Future research should focus on enhancing current diagnostic tools and exploring novel technologies such as contrast-enhanced ultrasonography and advanced MRI protocols [86].
Artificial intelligence and machine learning algorithms offer substantial potential for enhancing the diagnostic process [87]. AI-based image analysis can assist in detecting subtle signs of CSP on ultrasonography, potentially improving diagnostic accuracy and consistency, particularly among less experienced operators [88].

7.2 Novel Therapeutic Approaches

While current treatment options for CSP include medical, surgical, and minimally invasive interventions, there remains a need for novel therapeutic approaches that enhance treatment efficacy while minimizing risks [89]. Targeted drug delivery systems represent a promising area of investigation [90]. Nanoparticle-based delivery systems could be engineered to selectively transport therapeutic agents to the ectopic pregnancy site, maximizing local drug concentrations while minimizing

systemic exposure and toxicity [91].

Gene therapy and molecular-targeted interventions represent frontier areas that warrant exploration [92]. Therapies targeting specific molecular pathways involved in trophoblastic invasion and angiogenesis could potentially inhibit CSP progression without the need for invasive procedures [93].

7.3 Clinical Practice Recommendations and Research Priorities

Several recommendations can be proposed for clinical practice and future research to improve CSP management [41]. Standardized diagnostic protocols should be developed and implemented, incorporating state-of-the-art imaging modalities and clearly defined diagnostic criteria [94].

A multidisciplinary approach to CSP management should be promoted, involving collaboration among obstetricians, reproductive endocrinologists, interventional radiologists, surgeons, and other specialists [95]. Patient and healthcare provider education regarding CSP risk factors and early signs should be enhanced [4].

Longitudinal studies are needed to assess the long-term outcomes of various management strategies for CSP [96]. These studies should evaluate not only immediate treatment success but also subsequent fertility, pregnancy outcomes, and quality of life [97]. Clinical trials examining novel therapeutic approaches are essential for advancing CSP treatment [98].

Registry-based studies and large observational cohorts could provide valuable real-world evidence regarding CSP epidemiology, risk factors, and outcomes [99]. Investigation of preventive strategies represents an important research priority [100]. Studies examining optimal caesarean section surgical techniques, suture materials, and closure methods that minimize scar defect formation could potentially reduce future CSP risk [101].

Finally, research into the psychosocial impact of CSP and effective support interventions is needed [102]. Understanding the emotional and psychological consequences of CSP diagnosis and treatment can inform development of comprehensive support programs [103].

Conclusion

Caesarean scar ectopic pregnancy represents a serious and increasingly recognized complication of caesarean delivery, with potential for significant maternal morbidity and mortality. The rising incidence of CSP parallels the global increase in caesarean section rates, making this condition an important public health concern. Prompt diagnosis through high-resolution imaging, particularly transvaginal ultrasonography, is essential for preventing catastrophic complications.

Management of CSP requires individualized treatment planning based on patient factors, pregnancy characteristics, and available resources. Options ranging from expectant management to medical therapy to various surgical approaches allow for tailored treatment that considers both immediate safety and long-term fertility goals. Emerging technologies including uterine artery embolization and combined minimally invasive approaches have expanded the therapeutic armamentarium and improved outcomes.

Despite advances in diagnosis and treatment, CSP continues to pose significant challenges, and further research is needed to optimize management strategies and prevent complications. Development of standardized protocols, advancement of diagnostic technologies, investigation of novel therapeutic approaches, and comprehensive long-term outcome studies represent important priorities for future research. Through continued scientific investigation and clinical innovation, outcomes for women affected by CSP can be further improved (Figure 2).

Acknowledgement

This work was supported by Multidisciplinary Research Unit of SCB medical college and hospital, Cuttack, Odisha.

Table 1: Comparative Analysis of Diagnostic Methods for Cesarean Scar Ectopic Pregnancy (CSEP)

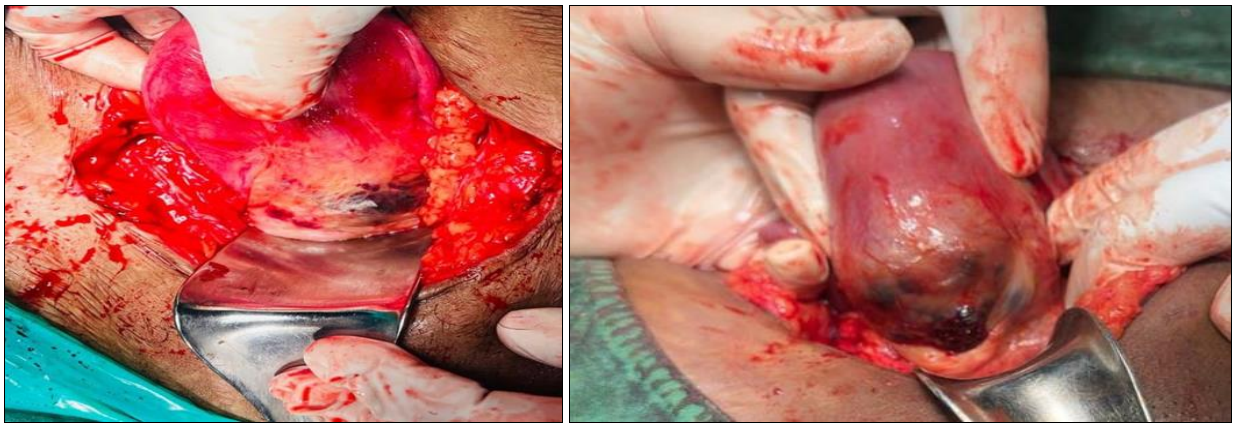
Diagnostic Method	Description	Advantages	Limitations	Key Studies
Transvaginal Ultrasonography (TVS)	Utilizes high-frequency sound waves to create detailed images of pelvic structures.	Non-invasive, widely available, high sensitivity for detecting gestational sac in scar tissue.	Operator-dependent, may be less effective in later pregnancy stages.	Monteagudo *et al.* (2001), Godin *et al.* (1997)
Magnetic Resonance Imaging (MRI)	Uses strong magnetic fields and radio waves to generate detailed images of organs and tissues.	Excellent soft tissue contrast, useful for detailed anatomical evaluation, non-ionizing.	Expensive, less accessible, time-consuming.	Sugawara *et al.* (2005), Vial *et al.* (2000)
Hysteroscopy	Involves the insertion of a hysteroscope through the cervix to directly visualize the uterine cavity.	Allows direct visualization and potential for therapeutic intervention.	Invasive, requires anaesthesia, potential for complications.	Pirtea *et al.* (2019), Rampen (1997)
3D Ultrasonography	Advanced ultrasound technique providing three-dimensional images of pelvic structures.	Improved spatial resolution, better visualization of scar and gestational sac relationship.	Limited availability, operator-dependent, may be more expensive than 2D ultrasound.	Calì *et al.* (2018), Xiao *et al.* (2014)
Contrast-Enhanced Ultrasonography	Enhances ultrasound imaging with the use of contrast agents to improve visualization.	Better delineation of blood flow and tissue structures, potential for enhanced diagnostic accuracy.	Requires administration of contrast agent, potential for allergic reactions.	Monteagudo *et al.* (2001), Godin *et al.* (1997)
Doppler Ultrasonography	Utilizes Doppler effect to assess blood flow within the pelvic vessels.	Non-invasive, useful for evaluating vascularization of the ectopic pregnancy.	Operator-dependent, less detailed anatomical information compared to MRI.	Vial *et al.* (2000), Jauniaux *et al.* (2021)
Serum Beta-hCG Measurement	Measures levels of beta-human chorionic gonadotropin in the blood.	Non-invasive, useful for initial diagnosis and monitoring treatment response.	Non-specific, cannot localize ectopic pregnancy on its own.	Jurkovic *et al.* (2003), Thakur and Shrimali (2023)
Endometrial Biopsy	Sampling of endometrial tissue to identify pregnancy location.	Can provide histological confirmation of ectopic pregnancy.	Invasive, not routinely used, potential for procedural complications.	Sugawara *et al.* (2005), Vial *et al.* (2000)
Combined TVS and MRI	Use of both ultrasound and MRI to enhance diagnostic accuracy.	Provides comprehensive anatomical details and vascular	Expensive, requires coordination between different	Monteagudo *et al.* (2001), Sugawara *et*

		information.	imaging modalities.	*al*. (2005)
Color Doppler Imaging	Uses color Doppler technology to visualize blood flow in and around the ectopic pregnancy.	Non-invasive, can assess vascularity, useful for surgical planning.	Operator-dependent, may not always distinguish between normal and abnormal blood flow.	Maymon *et al*. (2004), Jauniaux *et al*. (2021)
Saline Infusion Sonohysterography	Injects saline into the uterine cavity during ultrasound to enhance visualization of uterine lining.	Provides better contrast and clearer images of the uterine cavity.	Invasive, discomfort during procedure, risk of infection.	Monteagudo *et al*. (2001), Godin *et al*. (1997)
Serum Progesterone Levels	Measures progesterone levels to help differentiate between viable and non-viable pregnancies.	Non-invasive, useful adjunct to other diagnostic methods.	Non-specific, cannot localize ectopic pregnancy.	Jurkovic *et al*. (2003), Thakur and Shrimali (2023)
Ultrasound-Guided Aspiration	Uses ultrasound to guide a needle to aspirate the gestational sac for diagnostic confirmation.	Direct sampling, can provide immediate results.	Invasive, potential for bleeding and infection.	Sugawara *et al*. (2005), Vial *et al*. (2000)
Hysterosalpingography	X-ray technique using contrast dye to visualize the uterine cavity and fallopian tubes.	Can identify structural abnormalities, useful for patients with recurrent ectopic pregnancies.	Radiation exposure, discomfort, risk of allergic reaction to contrast dye.	Monteagudo *et al*. (2001), Sugawara *et al*. (2005)
Serum CA-125 Levels	Measures cancer antigen 125 levels which may be elevated in some ectopic pregnancies.	Non-invasive, potential early indicator.	Non-specific, not widely used, requires further validation.	Vial *et al*. (2000), Jauniaux *et al*. (2021)
CT Scan	Uses computed tomography to provide detailed cross-sectional images of the body.	High-resolution images, useful for complex cases.	High radiation exposure, expensive, less specific than MRI for soft tissues.	Sugawara *et al*. (2005), Vial *et al*. (2000)
Laparoscopy	Direct visualization of the pelvis through a small incision using a camera.	Definitive diagnosis, allows for immediate treatment.	Invasive, requires anaesthesia, potential for surgical complications.	Lee *et al*. (1999), Rampen (1997)
Bimanual Examination	Physical examination to assess the size and shape of the uterus and adnexa.	Non-invasive, can provide initial indication of ectopic pregnancy.	Less sensitive and specific, requires further imaging for confirmation.	Godin *et al*. (1997), Jurkovic *et al*. (2003)

Table 2: Overview of Therapeutic Approaches for Cesarean Scar Ectopic Pregnancy (CSEP)

Therapeutic Approach	Description	Indications	Success Rate	Complications	Key Studies
Methotrexate Therapy	Systemic or local administration of methotrexate to inhibit rapidly dividing cells in ectopic tissue.	Early-stage pregnancies, non-viable gestational sac, stable patients.	Variable	Potential for incomplete resolution, need for multiple doses, side effects.	Jurkovic *et al*. (2003), Ben Nagi *et al*. (2005)
Systemic Methotrexate	Intramuscular injection of methotrexate.	Non-viable gestational sac, small sac size, low beta-hCG levels.	70-80%	Gastrointestinal side effects, liver toxicity, bone marrow suppression.	Godin *et al*. (1997), Jurkovic *et al*. (2003)
Local Methotrexate Injection	Direct injection of methotrexate into the gestational sac under ultrasound guidance.	Viable gestational sac, higher beta-hCG levels, localized treatment.	80-90%	Local tissue necrosis, potential for infection, pain at injection site.	Ben Nagi *et al*. (2005), Pirtea *et al*. (2019)
Laparoscopic Surgery	Minimally invasive surgical removal of ectopic tissue using laparoscopic techniques.	Larger sac size, failed medical management, hemodynamic instability.	85-95%	Surgical risks (bleeding, infection), formation of adhesions.	Lee *et al*. (1999), Sugawara *et al*. (2005)
Hysteroscopic Surgery	Direct visualization and removal of ectopic tissue via a hysteroscope inserted through the cervix.	Small to medium sac size, localized ectopic tissue, desire to preserve fertility.	80-90%	Risk of uterine perforation, infection, anesthesia-related complications.	Rampen (1997), Vial *et al*. (2000)
Uterine Artery Embolisation (UAE)	Minimally invasive procedure to reduce blood supply to the ectopic tissue by embolizing uterine arteries.	Heavy bleeding, large or vascularized ectopic tissue, as an adjunct to other treatments.	75-85%	Ischemic pain, risk of non-target embolization, potential impact on future fertility.	Thakur and Shrimali (2023), Sugawara *et al*. (2005)
Combined Approach (Methotrexate + Surgery)	Combination of methotrexate therapy followed by surgical removal of ectopic tissue.	Complex cases, failed single modality treatment, recurrent cases.	90-95%	Combined risks of both medical and surgical interventions.	Jurkovic *et al*. (2003), Pirtea *et al*. (2019)
Expectant Management	Close monitoring without active intervention for selected stable cases with non-viable pregnancies.	Small sac size, declining beta-hCG levels, no significant symptoms.	Variable	Risk of sudden rupture, heavy bleeding requiring emergency surgery.	Bai *et al*. (2012), Timothy and Mirable (2020)
Balloon Tamponade	Use of balloon catheters to apply pressure and control bleeding in the uterine cavity.	Significant bleeding, as an adjunct to surgical removal of ectopic tissue.	80-90%	Discomfort, risk of balloon displacement, potential for infection.	Jurkovic *et al*. (2003), Monteagudo *et al*. (2001)
Gene Therapy	Experimental approach targeting specific genes involved in ectopic pregnancy implantation and growth.	Early-stage research, potential future application.	Not yet established	Unknown, requires extensive clinical trials.	Calì *et al*. (2018)

High-Intensity Focused Ultrasound (HIFU)	Non-invasive technique using focused ultrasound waves to ablate ectopic tissue.	Small to medium sac size, patients seeking non-surgical options.	75-85%	Potential for incomplete ablation, requires specialized equipment.	Xiao *et al*. (2014), Virdis *et al*. (2019)
Foley Catheter Placement	Insertion of a Foley catheter to tamponade the uterine cavity and control bleeding.	Severe bleeding, adjunct to other treatments.	70-80%	Discomfort, risk of infection, potential for displacement.	Godin *et al*. (1997), Jurkovic *et al*. (2003)
Conservative Surgery	Surgical removal of ectopic tissue with minimal disruption to uterine structure.	Early-stage pregnancies, desire to preserve fertility.	80-90%	Risk of incomplete removal, potential for recurrence.	Vial *et al*. (2000), Sugawara *et al*. (2005)
Dilatation and Curettage (D&C)	Surgical procedure to remove tissue from inside the uterus.	Small, non-viable gestational sacs, stable patients.	60-70%	Risk of uterine perforation, infection, incomplete removal.	Lee *et al*. (1999), Rampen (1997)
Uterine Repair Surgery	Surgical repair of the uterine scar to prevent future ectopic pregnancies.	Women with recurrent CSEP, significant uterine scar defects.	Variable	Major surgery, risk of adhesions, impact on future fertility.	Jurkovic *et al*. (2003), Ben Nagi *et al*. (2005)
Single-Dose Methotrexate Protocol	Administration of a single dose of methotrexate to treat ectopic pregnancy.	Early-stage pregnancies, small sac size, low beta-hCG levels.	70-80%	Potential need for additional doses, side effects similar to systemic methotrexate.	Godin *et al*. (1997), Sugawara *et al*. (2005)
Multidose Methotrexate Protocol	Multiple doses of methotrexate with leucovorin rescue to treat ectopic pregnancy.	Higher initial beta-hCG levels, larger sac size, failed single-dose treatment.	75-85%	Increased risk of side effects, requires close monitoring.	Jurkovic *et al*. (2003), Vial *et al*. (2000)
Ultrasound-Guided D&C	Use of ultrasound to guide dilatation and curettage for precise removal of ectopic tissue.	Early to mid-stage pregnancies, localized ectopic tissue.	70-80%	Potential for incomplete removal, procedural risks similar to D&C.	Ben Nagi *et al*. (2005), Pirtea *et al*. (2019)



(A) (B)

Fig 1: (A & B)-Intra operative finding of scar site ectopic pregnancy.

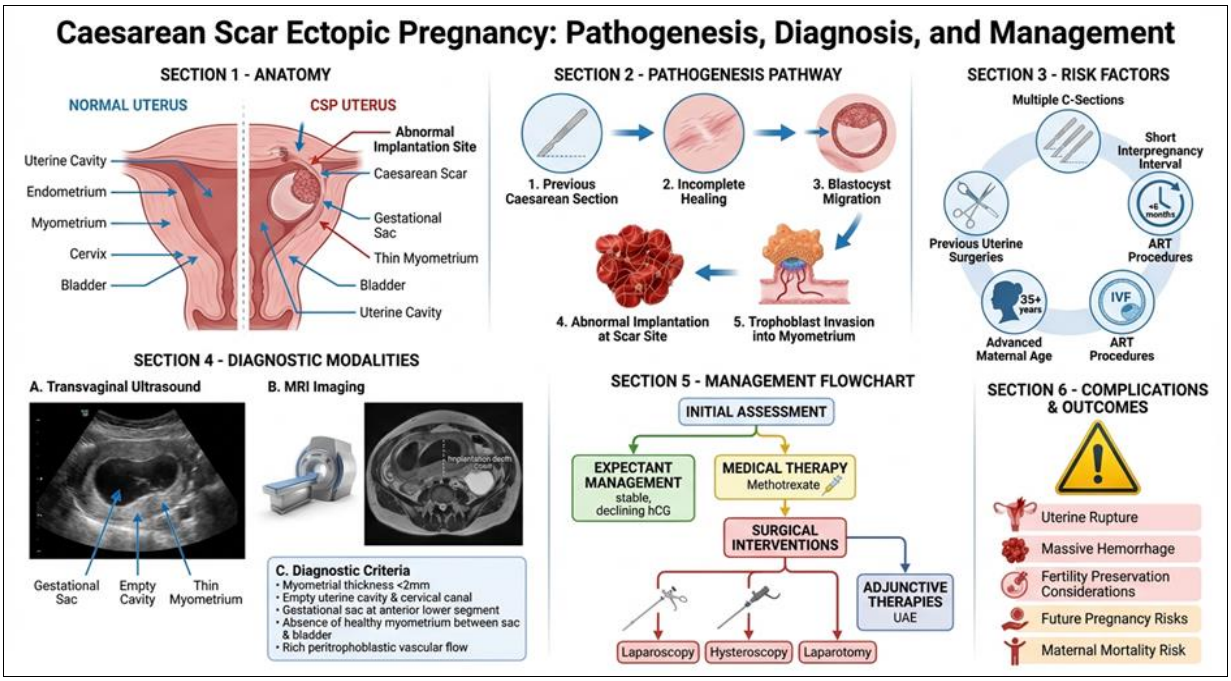


Fig 2: Overview of Caesarean Scar Ectopic Pregnancy: Pathogenesis, Diagnosis, and Management

Conflict of Interest
Not available

Financial Support
Not available

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How to Cite This Article
Jena M, Hembram M, Naik M. Caesarean scar ectopic pregnancy: Pathogenesis, diagnosis and management strategies. International Journal of Clinical Obstetrics and Gynaecology 2026; 10(1): 101-112.