International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Deraiya NA et al. Int J Reprod Contracept Obstet Gynecol. 2026 Dec;15(12):xxx-xxx
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20264248

Review Article

Maternal outcomes of infective endocarditis in pregnancy: a narrative review of diagnosis and multidisciplinary management

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Received: 21 October 2026
Accepted: 06 November 2026

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ABSTRACT

Infective endocarditis (IE) in pregnancy is rare (incidence about 0.006%) but increasing, driven by injection drug use, prosthetic valves and cardiac devices, and more women with congenital heart disease reaching reproductive age. Pregnancy obscures presentation and constrains diagnosis and treatment, and the evidence is scattered across case reports, small series and retrospective cohorts. This narrative review synthesizes published case reports, case series, cohort studies and reviews on IE in pregnancy and the postpartum period, focusing on maternal outcomes, risk factors, diagnosis and management, with fetal outcomes as a secondary consideration. Risk factors include injection drug use, rheumatic and congenital heart disease, prosthetic valves and intracardiac devices; *Staphylococcus aureus* predominates. Physiological murmurs, atypical symptoms and other febrile illnesses delay diagnosis. Contemporary maternal in-hospital mortality is 5-17% and, in the two largest comparative datasets, did not differ significantly from that of non-pregnant women. IE diagnosed during the delivery admission is the exception, with 17.2% mortality and severe morbidity in 77.5%. Heart failure, septic embolization and arrhythmia dominate morbidity. Valve surgery rates are similar to those in non-pregnant patients; bypass during pregnancy yields excellent maternal survival but 18-20% fetal mortality, favoring cesarean delivery followed by valve surgery once the fetus is viable. Multidisciplinary care, including addiction medicine, was associated with fewer patient-directed discharges. IE in pregnancy mainly alters recognition and management rather than survival of established disease, except at delivery. Early suspicion, echocardiography, blood cultures, pregnancy-compatible antimicrobials, timing of delivery and surgery, and tertiary multidisciplinary care are central. Prospective registries are needed.

Keywords: Infective endocarditis, Pregnancy, Maternal outcomes, Injection drug use, Cardiac surgery, Multidisciplinary care

INTRODUCTION

Infective endocarditis (IE) is a microbial infection of the endocardial surface, most often a cardiac valve.[1] Its global burden has risen markedly, with cases increasing from an estimated 478,000 in 1990 to over 1 million in 2019, and United States incidence from 10.2 to 14.4 per 100,000 population, with a disproportionate rise among women.[2,3] The opioid epidemic and injection drug use are leading contributors, alongside prosthetic valves, cardiovascular implantable electronic devices and healthcare-associated infection.[2,4,5] Congenital heart disease is a further, likely underrecognized contributor, as more women with structural heart disease reach reproductive age.[6] IE in pregnancy was historically considered exceedingly

uncommon, with an incidence of approximately 0.006%, although this figure appears to be rising.[3,7]

Pregnancy makes IE harder to detect and to manage. Increased cardiac output, expanded plasma volume and modulated maternal immunity lower resistance to infection and predispose women with valvular disease to hemodynamic decompensation.[1,4] Pregnancy-related hypercoagulability may raise embolic risk: emboli occur in about one quarter of IE cases in the general population but have been reported in up to half of pregnant patients.[8] Physiological flow murmurs can obscure a pathological murmur.[8] Once IE is suspected, antimicrobial and imaging choices must account for fetal toxicity, and the timing of surgery must balance maternal stability against the fetal risks of cardiopulmonary bypass.[3,4] Pregnancy makes IE harder to detect and to manage. Increased cardiac output, expanded plasma volume and modulated maternal immunity lower resistance to infection and predispose women with valvular disease to hemodynamic decompensation.[1,4] Pregnancy-related hypercoagulability may raise embolic risk: emboli occur in about one quarter of IE cases in the general population but have been reported in up to half of pregnant patients.[8] Physiological flow murmurs can obscure a pathological murmur.[8] Once IE is suspected, antimicrobial and imaging choices must account for fetal toxicity, and the timing of surgery must balance maternal stability against the fetal risks of cardiopulmonary bypass.[3,4]

The evidence base remains limited. Most data derive from non-pregnant populations, and pregnancy-specific maternal outcomes are dispersed across case reports, small series and retrospective cohorts.[1,2,6] Randomized trials in pregnancy are lacking, and the effects of maternal age, diagnostic delay and access to multidisciplinary care are poorly characterized.[9] This review synthesizes the available evidence on IE in pregnancy, with a primary focus on maternal outcomes and fetal outcomes as secondary context, to clarify risk factors and management challenges and to support earlier diagnosis and more standardized care.

METHODS

This narrative review synthesized published evidence on IE in pregnancy and the postpartum period, focusing on maternal outcomes, with fetal outcomes as secondary context. PubMed/MEDLINE, Embase, Scopus, and Google Scholar were searched from 2016 to 2026 using combinations of “infective endocarditis,” “endocarditis,” “pregnancy,” “postpartum,” “maternal outcomes,” “injection drug use,” and “cardiopulmonary bypass.” Case reports, case series, cohort studies, and reviews reporting pregnancy-specific data were included; general-population IE publications provided background only. Study design, population, risk factors, organisms, diagnosis, management, delivery, and outcomes were extracted and synthesized thematically. Large cohorts were prioritized for quantitative estimates; case reports illustrated management.

EPIDEMIOLOGY AND RISK FACTORS

IE is rare but life-threatening, with an incidence of 0.006% in pregnancy.[10] Pregnancy-associated IE is linked to maternal and fetal morbidity and mortality, including preterm birth, stillbirth and fetal death.[10,11] Incidence is rising, attributed to increasing intravenous drug and opioid use, long-term indwelling prosthetic cardiac devices and an aging population with multimorbidity; traditional risk factors such as immunosuppression, hemodialysis and prosthetic devices remain important.[1,2,9,10] Women with pregnancy-associated IE are generally younger than those with non-pregnancy-associated IE.[10] The opioid epidemic has been strongly linked to pregnancy-associated disease, and injection drug use during pregnancy is associated with infection-related complications, including IE.[1,12,13]

Established risk factors comprise rheumatic and congenital heart disease, prosthetic valves, hemodialysis, intravenous catheters and immunosuppression, while intracardiac devices and injection drug use are emerging contributors.[9] Among pregnant women with injection drug use–associated IE, tricuspid valve involvement is common even without pre-existing cardiac disease.[14] Procedures that cause bacteremia, such as epidural anesthesia, urinary catheterization, cesarean section and amniocentesis, add risk when aseptic technique is compromised, as do premature membrane rupture, prolonged labor and manual removal of the placenta.[1]

PATHOPHYSIOLOGY AND HEMODYNAMIC CONSIDERATIONS IN PREGNANCY

IE results from the interaction between host immunity and invading microorganisms and predominantly involves the cardiac valves, with a predilection for left-sided structures; the mitral valve is most commonly affected. Pregnancy-related increases in preload (about 50%), heart rate and cardiac output increase turbulence across the valves and injure the endothelium; platelet and fibrin deposition then provide a surface for microbial adherence and vegetation formation.[1,2,12] Fragmentation of vegetations may cause pulmonary embolism in right-sided IE and systemic embolism in left-sided IE.[9,11] In women with congenital heart disease, particularly bicuspid aortic valve, the same endothelial injury favors thrombus formation and subsequent infection.[9,12]

Cardiovascular adaptations of pregnancy may worsen or unmask cardiac disease.[7] Pregnancy has long been regarded as an immunosuppressed state, but this is an oversimplification: the maternal immune system is extensively reprogrammed, with enhanced innate responses and modulated adaptive immunity.[2,7,10] These adaptations protect the fetus but may reduce the capacity to clear invading pathogens, increasing susceptibility to infection, including IE.[1] Cardiovascular adaptations of

pregnancy may worsen or unmask cardiac disease.[7] Pregnancy has long been regarded as an immunosuppressed state, but this is an oversimplification: the maternal immune system is extensively reprogrammed, with enhanced innate responses and modulated adaptive immunity.[2,7,10] These adaptations protect the fetus but may reduce the capacity to clear invading pathogens, increasing susceptibility to infection, including IE.[1]

CLINICAL PRESENTATIONS AND DIAGNOSTIC CHALLENGES

Physiological adaptations of pregnancy, including increased preload and heart rate and decreased afterload, may mimic cardiovascular disease, and flow murmurs complicate examination. Presentation ranges from low-grade fever and non-specific symptoms to rapidly progressive disease.[1] Classical signs (fever, petechiae, Roth spots, Janeway lesions, Osler nodules, murmurs) may be absent or masked.[1,9] Persistent high fever is a warning sign, given the overlap with chorioamnionitis, pyelonephritis, pneumonia and viral infections; heart failure or embolization should prompt evaluation for IE.[1,9]

The modified duke criteria remain the primary diagnostic framework, and according to the American heart association the approach in pregnancy is similar to that in non-pregnant patients.[1,2] Blood culture is among the most reliable tests, and three sets before antibiotics are recommended.[1] Plasma microbial cell-free DNA sequencing may help establish culture-negative IE, but its performance depends on pathogen burden and the timing of antimicrobial exposure, so it should be interpreted alongside Duke criteria, microbiology and imaging.[9,15]

Transthoracic echocardiography (TTE) is the most common imaging modality; it is safe in pregnancy and can identify prognostic indicators such as systolic dysfunction, a subaortic gradient above 30 mmHg and pulmonary artery systolic pressure above 50 mmHg without right ventricular outflow obstruction.[1,9] Transesophageal echocardiography (TEE) is more sensitive and slightly more specific, particularly for vegetations under 5 mm, prosthetic valve IE or abscesses, but carries risks of emesis, aspiration and sedation-related complications.[1,9,16] FDG-PET/CT is gaining attention as an adjunct, but fetal radiation exposure requires careful consideration.[5] TTE is the most common imaging modality; it is safe in pregnancy and can identify prognostic indicators such as systolic dysfunction, a subaortic gradient above 30 mmHg and pulmonary artery systolic pressure above 50 mmHg without right ventricular outflow obstruction.[1,9] TEE is more sensitive and slightly more specific, particularly for vegetations under 5 mm, prosthetic valve IE or abscesses, but carries risks of emesis, aspiration and sedation-related complications.[1,9,16] FDG-PET/CT is gaining attention as an adjunct, but fetal radiation exposure requires careful consideration.[5]

A negative echocardiogram does not exclude IE, so diagnosis should integrate the clinical picture and a multidisciplinary approach involving cardiologists, surgeons, infectious disease specialists, obstetricians and neonatologists.[2,9,17] Evaluation is warranted in pregnant women with fever, unexplained tachycardia, a new murmur or signs of septic emboli.[1]

MICROBIOLOGY

Staphylococcus aureus is the predominant organism in pregnant and non-pregnant women, followed by polymicrobial infection, streptococci and enterococci, although non-pregnancy-associated IE shows greater microbial diversity.[4,10] Frequent healthcare exposure in pregnancy is a potential risk for healthcare-associated infection, particularly MRSA, and rising staphylococcal IE has been linked to injection drug use, prosthetic valves and cardiac devices.[2,13] S. aureus infection carries a poor prognosis; in one series of injection drug use–associated IE in pregnancy, MSSA accounted for 40% of cases and MRSA for 20%.[1,9,14]

Viridans streptococci, part of normal oral flora, may cause IE after dental procedures. Fungal involvement, most often Candida followed by Aspergillus, should be considered; zygomycetes have been reported with prolonged antimicrobial therapy and indwelling catheters.[1,9] Screening for streptococcus agalactiae has reduced beta-hemolytic streptococcal infection.[2] Culture-negative IE remains a challenge, and PCR, histopathology of resected valves and cell-free DNA sequencing may improve pathogen identification.[9]

MATERNAL OUTCOMES

Reported maternal mortality in IE in pregnancy varies widely with era, case mix and access to care: early estimates were as high as 33%, recent reviews about 11%, and contemporary series 5-17.2%.[1] In the largest analysis, a national database of 382 maternity-associated IE hospitalizations, in-hospital mortality was 8.1%, not significantly different from 10.6% in non-maternity-associated women of reproductive age (adjusted rate ratio [aRR] 1.03, 95% confidence interval [CI] 0.71-1.48).[10] A single-center review of 34 pregnant or postpartum patients likewise found in-hospital mortality of 2.9% versus 9.6% in matched non-pregnant patients, with no significant difference (Table 1).[4] Pregnancy itself therefore does not appear to increase mortality once IE has developed; outcome is driven by valve destruction, the pathogen and the timeliness of treatment. Figure 1 compares in-hospital mortality in these datasets and when IE complicates the delivery admission.

Outcomes are markedly worse when IE complicates the delivery admission itself: in-hospital mortality was 17.2% (aRR 323.32, 95% CI 127.74-818.37), severe maternal morbidity 77.5% (aRR 47.06, 95% CI 38.10-58.14) and cesarean delivery 56.1%.[10] These large ratios reflect a

near-zero baseline risk in uncomplicated deliveries and signal severity rather than a literal multiplier.

Morbidity is dominated by heart failure, embolic events, septic shock and arrhythmia. Thromboembolic events occurred in 54.7% of maternity-associated cases in the national cohort, less often than in non-maternity-associated IE after adjustment (aRR 0.73, 95% CI 0.63-0.84).[10] Septic cerebral embolization is a particular concern: in a six-patient multidisciplinary series, all with injection drug use, five needed intensive care and ventilation and three had cerebral emboli, including two strokes and one intracranial hemorrhage.[12] Embolic stroke has been reported in up to 30% of cases in some series, and ruptured mycotic aneurysms can cause fatal hemorrhage.[1,18]

Comparisons of medical and surgical management are limited by selection bias, since surgical candidates have the most severe disease, but valve surgery appears to carry acceptable maternal risk. Valve procedures were performed in 15.5% of maternity-associated versus 15.8% of non-maternity-associated admissions.[10] In the single-center review, replacement and repair rates were similar to those in non-pregnant patients, and every operation in pregnant patients was performed postpartum; in the Pittsburgh series, four of six patients had valve replacement, all after delivery or miscarriage, with no deaths at 90 days.[4,12]

Surgery during pregnancy remains high-risk for the fetus: among 22 women undergoing cardiopulmonary bypass (CPB), maternal mortality was 0% but fetal mortality was 18.2%.[19] Case reports illustrate the range of approaches: cesarean delivery at 33 weeks followed four days later by aortic valve replacement for bicuspid valve endocarditis with annular abscess and mycotic aneurysms, with a good outcome at six months, and valve replacement at 24 weeks in a woman with cerebral embolism.[18,20] In a Chinese series of four patients operated on under CPB during pregnancy, all mothers and fetuses survived.[21] Surgery during pregnancy remains high-risk for the fetus: among 22 women undergoing CPB, maternal mortality was 0% but fetal mortality was 18.2%.[19] Case reports illustrate the range of approaches: cesarean delivery at 33 weeks followed four days later by aortic valve replacement for bicuspid valve endocarditis with annular abscess and mycotic aneurysms, with a good outcome at six months, and valve replacement at 24 weeks in a woman with cerebral embolism.[18,20] In a Chinese series of four patients operated on under CPB during pregnancy, all mothers and fetuses survived.[21]

The principal cohort studies and case series informing these outcomes are summarized in Table 1.

Mode and timing of delivery require individualized planning. Cesarean delivery was more frequent when IE complicated the delivery admission (56.1% versus 32.2%, aRR 2.04).[10] A two-step strategy of cesarean delivery followed by cardiac surgery days later has been used successfully and aims to avoid catastrophic uterine hemorrhage when delivery and bypass occur in close succession.[11,18] When the fetus is previable or maternal condition permits, continuing the pregnancy under close surveillance until viability, then delivery followed by cardiac surgery, is increasingly favored.[11]

Postpartum outcomes and relapse risk are shaped by the underlying risk factors. Among patients with injection drug use-associated IE, relapse and recurrent IE are common: 30-day readmission exceeded 20% and relapsed injection drug use occurred in 45.2% of pregnant patients versus 51.7% of non-pregnant patients in the larger single-center review, and two of six patients in the Pittsburgh series developed recurrent IE.[4,12] Conversely, with a multidisciplinary team no patient-directed discharges occurred and five of six patients left on medication for opioid use disorder.[12] Maternal outcome therefore depends on addiction care, social support and contraception planning as well as antimicrobial and surgical decisions.

Postpartum IE can also arise through distinct mechanisms: Enterococcus faecalis mitral valve endocarditis three months after cesarean delivery in a woman without conventional risk factors; polymicrobial prosthetic valve endocarditis two weeks after a preterm home delivery in a woman with prior injection drug use; and Abiotrophia defectiva mitral valve endocarditis on postpartum day nine in a woman with orthodontic braces.[22-24]

MANAGEMENT STRATEGIES

Antimicrobial therapy follows the principles used in non-pregnant patients, with a duration of 4-6 weeks for native and 6 weeks for prosthetic valve infection, but fetal toxicity narrows the options.[1] Increased renal clearance may require higher penicillin doses or shorter dosing intervals.[1] β-lactams, vancomycin, daptomycin and clindamycin are generally considered compatible with all trimesters, whereas aminoglycosides and tetracyclines should be reserved for critical situations because of fetal ototoxicity and dental or osseous toxicity, respectively.[1] Pathogen-directed options are summarized in Table 2. Empirical therapy should start promptly after three sets of blood cultures and cover staphylococci, streptococci and enterococci; because vancomycin is generally avoided in pregnancy, daptomycin plus ceftriaxone or cefazolin is a reasonable alternative.[1] In documented penicillin allergy, skin testing and desensitization may allow use of the preferred agent.[25] Antimicrobial therapy follows the principles used in non-pregnant patients, with a duration of 4-6 weeks for native and 6 weeks for prosthetic valve infection, but fetal toxicity narrows the options.[1] Increased renal clearance may require higher penicillin doses or shorter dosing intervals.[1] β-lactams, vancomycin, daptomycin and clindamycin are generally considered compatible with all trimesters, whereas aminoglycosides and tetracyclines should be reserved for critical situations because of fetal ototoxicity and dental or osseous toxicity,

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Indications for surgery in pregnancy mirror those in non-pregnant patients: heart failure from acute regurgitation or cardiogenic shock is an absolute indication, whereas uncontrolled infection and prevention of embolism are relative indications requiring individualized assessment, with the threshold weighed against the fetal risk of CPB.[1] CPB is associated with uterine contraction, placental hypoperfusion and fetal hypoxia, and fetal mortality remains about 20% in contemporary series, with cooling and rewarming the most vulnerable periods.[19,21] Proposed mitigation includes normothermia or mild hypothermia (above 34°C), pulsatile high-flow, high-pressure perfusion, avoidance of hemodilution, short bypass and cross-clamp times, continuous fetal heart rate monitoring and perioperative tocolysis.[19-21] When the fetus is viable, delivery before cardiac surgery is generally preferred, and a two-stage approach has been used successfully.[11,18]

Multidisciplinary care is the most consistent theme in the literature. The European society of cardiology recommends that pregnant patients with IE be managed in tertiary centers by a dedicated team.[1] The Pittsburgh endocarditis team, spanning infectious diseases, cardiology, cardiac surgery, neurology, psychiatry, addiction medicine and obstetrics, achieved a high rate of surgical intervention, no patient-directed discharges and no in-hospital or 90-day mortality among six patients with injection drug use-associated S. aureus IE.[12] The Chinese series likewise attributed its success to team formation and individualized perioperative management.[21] Delivery planning should address mode and timing of delivery relative to cardiac surgery, anticoagulation bridging for mechanical prostheses, and antibiotic prophylaxis within one hour before incision for high-risk patients undergoing invasive procedures.[1]

FETAL AND NEONATAL OUTCOMES

Fetal and neonatal outcomes are poor, driven by maternal hemodynamic compromise and the consequences of preterm delivery. Preterm birth is the most consistently reported adverse outcome, occurring in 46-55.7% of pregnancies with mean gestational ages at delivery of 32.4-35.0 weeks.[4,10] In the national cohort, delivery complicated by IE carried higher risks of preterm birth (55.7% versus 10.1%, aRR 3.61, 95% CI 2.56-5.08) and stillbirth (aRR 2.96, 95% CI 1.21-7.24).[10] Case reports describe favorable neonatal outcomes after preterm delivery at 31-33 weeks.[11,18,24] These outcomes reflect maternal disease severity and iatrogenic preterm delivery, so timely maternal diagnosis and treatment is the most effective strategy for improving neonatal outcomes.

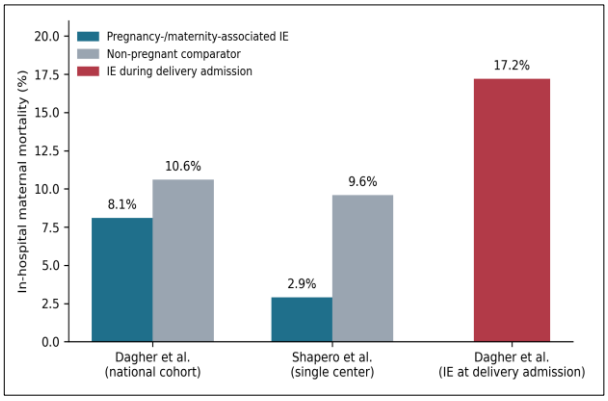


Figure 1: In-hospital maternal mortality in infective endocarditis (IE). Bars compare women with pregnancy- or maternity-associated IE with non-pregnant comparators in the national cohort of Dagher et al and the single-center study of Shapero et al, and show mortality when IE complicated the delivery admission itself. The authors reported no significant difference between pregnant and comparator groups in either dataset; the delivery-admission estimate reflects a near-zero baseline risk in uncomplicated deliveries and is best read as a marker of severity.[4,10]

Table 1: Principal cohort studies and case series informing maternal and fetal outcomes of infective endocarditis in pregnancy.

Study	Setting and design	Population	Maternal outcomes	Fetal and neonatal outcomes
Dagher et al[10]	United States; retrospective national cohort (national readmissions database)	382 maternity-associated IE hospitalizations (weighted national estimate 748)	In-hospital mortality 8.1% versus 10.6% in non-maternity-associated women of reproductive age (aRR 1.03, 95% CI 0.71-1.48). IE at delivery admission: mortality 17.2%, severe maternal morbidity 77.5%. Thromboembolic events 54.7%	Preterm birth 55.7% versus 10.1% (aRR 3.61); stillbirth aRR 2.96

Continued.

Study	Setting and design	Population	Maternal outcomes	Fetal and neonatal outcomes
Shapero et al[4]	United States (Pittsburgh); single-center retrospective chart review with matched non-pregnant comparators	34 pregnant or postpartum patients	In-hospital mortality 2.9% versus 9.6%; 1- and 2-year mortality about 15% and 20.6%, not significantly different. Valve replacement 32.4% versus 29.5%; all operations performed postpartum. Relapsed injection drug use 45.2%	Perinatal mortality 19.2%; preterm delivery 50%; NICU admission 68%
Shapero et al[12]	United States (Pittsburgh); multidisciplinary case series	6 patients, all with injection drug use-associated *S. aureus* IE	No in-hospital or 90-day mortality. Valve replacement in 4 of 6, all after delivery or miscarriage. No patient-directed discharges; 5 of 6 discharged on medication for opioid use disorder; 2 of 6 recurrent IE	4 live births (mean gestational age 32.4 weeks); 3 NICU admissions; 1 miscarriage at 10 weeks
Dahshan et al[14]	United States (West Virginia); case series	10 pregnant patients with injection drug use-associated IE	Septic embolization 40%; hemorrhagic stroke 10%; no maternal deaths during admission (follow-up incomplete).	Fetal loss 30% (11-22.5 weeks)
Liu et al[19]	China; case series of cardiac operations under CPB during pregnancy*	22 pregnant women	Maternal mortality 0%; atrial fibrillation the most common complication (13.6%)	Fetal mortality 18.2% (4 intrauterine deaths, 2 neonatal deaths from intracranial hemorrhage, 3 terminations)
You et al[21]	China; case series of cardiac surgery under CPB during pregnancy*	4 pregnant women (3 with IE, 1 with ascending aortic aneurysm)	All 4 mothers survived; 2 underwent simultaneous cesarean section and cardiac surgery and 2 continued pregnancy.	All 4 fetuses survived

aRR- adjusted rate ratio; CI- confidence interval; CPB- cardiopulmonary bypass; IE- infective endocarditis; NICU- neonatal intensive care unit. *Cardiac surgery series that were not limited to IE. The series of Shapero et al and originate from the same institution and may include overlapping patients.[4,12]

Table 2: Pathogen-directed antimicrobial options for infective endocarditis in pregnancy.

Scenario	Regimens reported	Pregnancy-specific considerations
Penicillin-susceptible viridans group streptococci and S. gallolyticus	Penicillin G, amoxicillin, or ceftriaxone for 4-6 weeks	β-Lactams are generally compatible with all trimesters; increased renal clearance may require higher doses or shorter dosing intervals for penicillins
Penicillin-resistant *Streptococci*	Daptomycin combined with ceftriaxone or ceftaroline has been proposed	Standard regimens incorporating gentamicin or vancomycin are problematic in pregnancy; pregnancy-specific efficacy data are limited
Methicillin-susceptible *S. aureus* (native or prosthetic valve)	Native valve: cloxacillin, flucloxacillin, cefazolin, or cefotaxime. Prosthetic valve: daptomycin plus ceftaroline or fosfomycin preferred	Daptomycin is generally considered compatible with all trimesters
Methicillin-resistant *S. aureus*, prosthetic valve	Standard rifampicin- and gentamicin-containing regimens	Fetal risk; individualized risk-benefit assessment required
Enterococci	Ampicillin or amoxicillin plus ceftriaxone for 6 weeks; daptomycin-based combinations for multidrug-resistant strains	Aminoglycosides avoided where possible
Empirical therapy (suspected native or prosthetic valve IE)	Start promptly after three sets of blood cultures at 30-minute intervals; cover staphylococci, streptococci, and enterococci; daptomycin plus ceftriaxone	Vancomycin is generally avoided in pregnancy according to the source guidance[1]

Continued.

Scenario	Regimens reported	Pregnancy-specific considerations
	or cefazolin is a pregnancy-compatible alternative	
Fungal endocarditis	Liposomal amphotericin B with early surgery and multidisciplinary input	Mortality exceeds 50%
Agents to reserve for critical situations	Aminoglycosides; tetracyclines	Fetal ototoxicity (aminoglycosides); dental and osseous toxicity (tetracyclines)
Documented penicillin allergy	Skin testing and desensitization may allow use of the preferred agent[25]	Illustrated by a pregnant patient with Streptobacillus moniliformis endocarditis transitioned to high-dose penicillin G

Source: Polyzou et al, unless otherwise stated.[1] Total duration is 4-6 weeks for native valve and 6 weeks for prosthetic valve infection. IE, infective endocarditis. Regimens must be individualized; many pregnancy-compatible alternatives rest on limited pregnancy-specific evidence

DISCUSSION

This review consolidated the evidence on IE in pregnancy, focusing on maternal outcomes. Three themes emerge: pregnancy alters presentation and diagnosis more than survival; the peripartum period is the point of greatest danger; and outcomes depend on coordinated care rather than any single decision.

Diagnosis is difficult because hemodynamic adaptations, flow murmurs and overlap with common febrile illnesses can mask classical features, and delay matters given the high embolic burden.[1,4,9,10,12,14] A negative echocardiogram should not end the work-up when suspicion persists; repeat imaging and cell-free DNA sequencing may help in culture-negative disease.[9,15] Fever, unexplained tachycardia, a new murmur, heart failure or an embolic event in a woman with a cardiac substrate or injection drug use should prompt early evaluation.[1]

Mortality is lower than early reports suggested (from as high as 33% to 5-17%) and, in the two largest comparative datasets, did not differ significantly from that of non-pregnant women with IE; outcome appears driven by pathogen virulence, valve destruction and timeliness of treatment.[1,4,10] The exception is IE diagnosed during the delivery admission, with in-hospital mortality of 17.2% and severe maternal morbidity in over three-quarters of patients; the large adjusted ratios mark severity rather than a literal multiplier.[10] Peripartum IE should be treated as an obstetric emergency requiring immediate multidisciplinary mobilization.

Management is where pregnancy imposes its most distinctive constraints. Antimicrobial choice must balance efficacy against fetal toxicity, and many compatible alternatives, such as daptomycin-based combinations, rest on limited pregnancy-specific evidence.[1] Valve surgery rates were similar in pregnant and non-pregnant patients, and in the largest single-center experience every operation was performed postpartum without excess complications.[4,10] Bypass during pregnancy yields excellent maternal survival but fetal mortality of 18-20%, favoring cesarean delivery followed by valve surgery once the fetus is viable.[11,18,19,21] Management is where pregnancy imposes its most distinctive constraints.

Antimicrobial choice must balance efficacy against fetal toxicity, and many compatible alternatives, such as daptomycin-based combinations, rest on limited pregnancy-specific evidence.[1] Valve surgery rates were similar in pregnant and non-pregnant patients, and in the largest single-center experience every operation was performed postpartum without excess complications.[4,10] Bypass during pregnancy yields excellent maternal survival but fetal mortality of 18-20%, favoring cesarean delivery followed by valve surgery once the fetus is viable.[11,18,19,21]

Injection drug use is a leading and growing driver of pregnancy-associated IE, with frequent relapse and readmission.[1,4,12] When addiction medicine, psychiatry and social support joined the endocarditis team, patient-directed discharges were eliminated and most patients left on medication for opioid use disorder; maternal outcome therefore extends to substance use treatment and postpartum follow-up.[12]

Limitations

The evidence is dominated by case reports, small series, and single-center cohorts, all vulnerable to publication and selection bias. Administrative datasets lack microbiological, echocardiographic, and timing details and likely under-ascertain embolic events.[10] Comparison groups differ, follow-up is short, populations are heterogeneous, and recommendations are largely extrapolated from non-pregnant guidance. This narrative review cannot provide pooled estimates.

Future directions

Priorities include prospective multicenter registries with standardized outcome definitions, pharmacokinetic studies of key antimicrobials, evaluation of structured endocarditis team models, and studies of surgical timing, perioperative fetal protection, and long-term maternal outcomes.

CONCLUSION

Infective endocarditis in pregnancy is rare but potentially devastating, and its incidence is rising. Pregnancy

obscures its clinical presentation, so a high index of suspicion is essential. Once IE is established, maternal mortality does not appear to exceed that of non-pregnant patients, but the delivery admission carries a markedly higher risk of death and serious morbidity. Early echocardiography and blood cultures, pregnancy-compatible antimicrobial therapy, individualized timing of delivery and cardiac surgery, and multidisciplinary care at a tertiary center are the foundations of management, with addiction treatment and postpartum support integral for patients who inject drugs. Prospective data are needed to move practice toward standardized, evidence-based guidance.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Deraiya NA, Pathan S, Selwyn SR, Arumugam S, Wagh Y, Kongasseri B. Maternal outcomes of infective endocarditis in pregnancy: a narrative review of diagnosis and multidisciplinary management. Int J Reprod Contracept Obstet Gynecol 2026;15:xxx-xx.