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When Pregnancy Reveals a Hidden Heart Defect: Atrial Septal Defect in Severe Preeclampsia and Fetal Death - A Case Report

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Abstract

Congenital heart disease (CHD), particularly atrial septal defect (ASD), may remain clinically silent into adulthood and become unmasked only when the hemodynamic burden of pregnancy exceeds cardiac reserve. We report a 34-year-old primigravida with untreated pre-existing chronic hypertension who presented at 31 weeks of gestation with preterm prelabor rupture of membranes and uterine contractions. On admission, blood pressure was 189/118 mmHg with severe features of preeclampsia, and intrauterine fetal death (IUFD) was subsequently confirmed. She was managed with magnesium sulfate and oral nifedipine, and proceeded to spontaneous vaginal delivery. Because hypertension remained refractory even after delivery, cardiology consultation was requested, and postpartum echocardiography revealed a 12 mm secundum ASD with left-to-right shunt and right heart volume overload, without pulmonary hypertension. In this case, the ASD was recognized only after a stillbirth, prompted by refractory postpartum hypertension rather than by antenatal cardiac symptoms. We highlight that Indonesia's congenital heart disease screening strategy is directed at neonates rather than pregnant women, leaving maternal structural heart disease undetected until pregnancy-related complications occur. We argue for incorporation of basic cardiac screening into antenatal care and a low threshold for echocardiographic referral in women with refractory or atypical hypertension.

Keywords: Antenatal screening; atrial septal defect; congenital heart disease; intrauterine fetal death; maternal cardiology; preeclampsia; pregnancy

Introduction

Atrial septal defect (ASD) is the congenital heart disease (CHD) most commonly found in the adult population and often remains asymptomatic for years [1]. In small-to-moderate defects, the left-to-right shunt causes volume overload of the right atrium and right ventricle that develops gradually, so that structural and functional cardiac changes progress slowly without producing significant complaints [1,2]. Symptoms typically emerge only when the hemodynamic load increases, including during pregnancy, which physiologically raises cardiac output by approximately 40-50% together with a fall in systemic vascular resistance, and can thereby unmask a previously undiagnosed ASD [3]. Contemporary guideline-based clinical reviews similarly emphasize that ASD-related right heart overload may first become apparent only when the cumulative hemodynamic demands of pregnancy are superimposed on a previously well-compensated defect [4].

Atrial septal defect (ASD) that remains undetected during pregnancy may contribute to worse maternal and perinatal outcomes, as hemodynamics changes during gestation can increase the volume load on the right heart and raise the risk of complications in women with CHD [5]. Population-based cohort studies have shown that pregnancy complicated by CHD is associated with an increased risk of preeclampsia (adjusted OR 1.38; 95% CI 1.28-1.49), and is also linked to a higher incidence of fetal growth restriction (IUGR) and unfavorable perinatal outcomes [5,6]. A nationwide Norwegian cohort study corroborates this pattern, reporting higher rates of preeclampsia and adverse neonatal outcomes among pregnancies complicated by maternal CHD compared with pregnancies in structurally normal hearts [7]. However, this relationship appears lesion-dependent rather than uniform: a large Swedish register-based study found that pregestational heart failure as a whole was not independently associated with increased preeclampsia risk, whereas valvular heart disease specifically was, suggesting that the anatomical and hemodynamic characteristics of the underlying cardiac lesion-rather than the mere presence of heart disease-likely determine obstetric risk [8].

In Indonesia, the burden of CHD remains considerable, with an estimated incidence of approximately 8 cases per 1,000 live births, translating to roughly 50,000 infants born with CHD each year, of whom approximately 12,500 have critical CHD requiring early intervention [9]. Nevertheless, the national screening system, expanded since 2023, still focuses on critical congenital heart disease (CCHD) screening in neonates using pulse oximetry. Current evidence indicates that implementation of this screening in Indonesia remains limited to feasibility studies and pilot programs, and has not yet become part of universal national screening [9,10]. This pattern is not unique to Indonesia: pulse oximetry-based CCHD screening has been prioritized globally because it is inexpensive and simple to implement, yet it remains, by design, a neonate-directed strategy that offers no pathway for identifying non-cyanotic structural lesions in adults [11]. To date, no cardiac screening pathway has been integrated into routine antenatal care (ANC) for adult women, and current WHO ANC standards likewise do not mandate systematic cardiac auscultation or echocardiographic screening as part of routine antenatal contacts, leaving maternal structural heart disease to be identified only opportunistically [12]. This creates a gap in early detection, as adults with previously undiagnosed ASD are not routinely covered by systematic cardiovascular screening [4].

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This limitation is particularly relevant for ASD, as pulse oximetry primarily detects congenital heart defects associated with hypoxemia and may not identify lesions without significant oxygen desaturation. An ASD with a left-to-right shunt and normal pulmonary arterial pressure may therefore remain clinically silent and undetected by oxygen saturation-based screening. Furthermore, the absence of routine maternal echocardiography may limit the detection of mild or asymptomatic structural heart disease during pregnancy. Consequently, previously undiagnosed ASD may only be recognized when clinical findings raise suspicion of an underlying cardiac condition. When cardiovascular disease is suspected during pregnancy, electrocardiography is used to assess new cardiac signs or symptoms or suspected arrhythmia, while transthoracic echocardiography is the first-line cardiac imaging modality [4]. The consequences of such delayed recognition are not merely theoretical: stillbirth remains a substantial and often under-prioritized burden in low- and middle-income countries, where preventable causes-including undetected maternal cardiovascular and hypertensive disease-continue to be inadequately addressed by existing antenatal care systems [13].

This case report aims to present a pregnant woman with a previously undiagnosed ASD, identified only after she developed severe pre-eclampsia complicated by intrauterine fetal death (IUFD). This case illustrates the potential for limited cardiovascular screening in adults to delay the recognition of previously undiagnosed CHD and underscores the importance of clinical vigilance for underlying cardiac abnormalities during antenatal care.

Case Report

A 34-year-old primigravida presented at 31 weeks of gestation with preterm prelabor rupture of membranes accompanied by uterine contractions. She had attended antenatal care eight times at a community health center (three visits in the first trimester, two in the second, and three in the third) and had been noted to have elevated blood pressure from early pregnancy. On further history, she reported that her blood pressure had also been elevated before conception, but she had never sought medical evaluation or treatment because she was asymptomatic. She denied any cardiovascular symptoms - dyspnea, chest pain, palpitations, syncope, or easy fatigability - at any point, and no cardiac examination had previously been performed during antenatal visits. There was no family history of congenital or other heart disease, no history of rheumatic fever, and no history of diabetes, renal disease, or thyroid disorder. She did not smoke or consume alcohol, and this was her first pregnancy.

On admission, blood pressure was 189/118 mmHg, heart rate 109 beats/minute, respiratory rate 22 breaths/minute, oxygen saturation 95% on room air, and temperature 36.8 °C. Examination revealed bilateral lower-limb edema and epigastric tenderness. Obstetric examination showed a fundal height of approximately 31 cm, a single live fetus in longitudinal lie with cephalic presentation, and a closed cervix. Fetal heart rate was initially 112 beats/minute and regular; however, on subsequent observation fetal heart activity could no longer be detected, and ultrasonography confirmed absence of cardiac activity, establishing the diagnosis of intrauterine fetal death (IUFD). Laboratory investigations on admission showed urine protein 3+, hemoglobin 10.8 g/dL, leukocytes 12,600/µL, platelets 98,000/µL, AST 86 U/L, ALT 72 U/L, urea 42 mg/dL, creatinine 1.2 mg/dL, and uric acid 8.1 mg/dL; coagulation studies were within normal limits. These findings were consistent with chronic hypertension with superimposed preeclampsia with severe features, without evidence of disseminated intravascular coagulation.

A posteroanterior chest radiograph obtained on admission (Figure 1) demonstrated increased and hazy bronchovascular markings with a positive hilar haze sign and a cardiothoracic ratio exceeding 50%, an appearance consistent with early pulmonary edema. A radiographic suspicion of pericardial effusion was also raised on this film, although this finding was not subsequently confirmed onechocardiography.

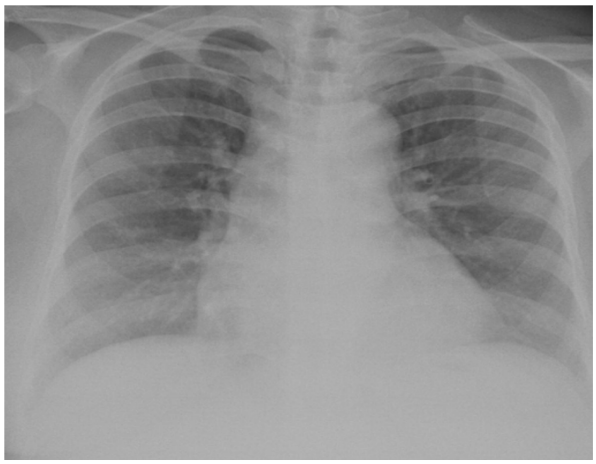

Figure 1: *Chest radiograph (posteroanterior view) on admission, showing increased and hazy bronchovascular markings, a positive hilar haze sign, and a cardiothoracic ratio greater than 50%, consistent with an early pulmonary edema pattern.*

Electrocardiography showed sinus rhythm at 96 beats/minute, right axis deviation, and incomplete right bundle branch block with an rSr' pattern in leads V1–V2, suggestive of right-sided volume overload (Figure 2).

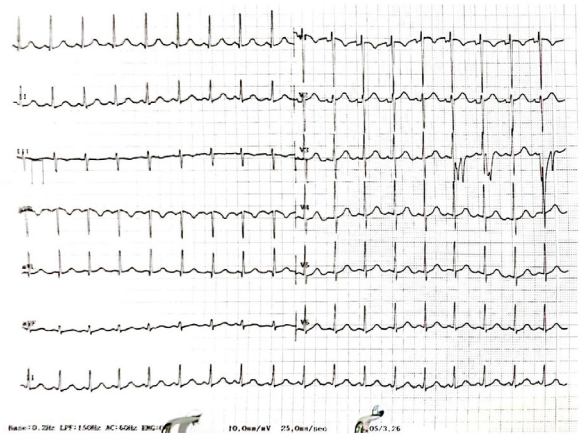

Figure 2: *Twelve-lead electrocardiogram obtained on admission, showing sinus rhythm at 96 beats/minute with right axis deviation and an incomplete right bundle branch block pattern (rSr') in leads V1–V2, findings suggestive of right heart volume overload. Paper speed 25 mm/s; calibration 10 mm/mV.*

The patient received magnesium sulfate for seizure prophylaxis (4 g intravenous loading dose, followed by 1–2 g/hour continuous intravenous infusion) and nifedipine 10 mg orally three times daily for blood pressure control. Fluid administration was restricted, and close hemodynamic monitoring was maintained. She subsequently underwent spontaneous vaginal delivery of a macerated female fetus. Because blood pressure remained persistently elevated and poorly responsive despite antihypertensive therapy, including after delivery, the obstetric team requested cardiology consultation to evaluate for an underlying cardiac cause. Postpartum echocardiography revealed situs solitus with atrioventricular and ventriculoarterial concordance, and a secundum atrial septal defect measuring approximately 12 mm with a left-to-right shunt. There was dilatation of the right atrium and right ventricle with preserved right ventricular systolic function, as demonstrated by a normal TAPSE, and preserved left ventricular sys-

tolic function (LVEF approximately 65%). Estimated pulmonary artery pressure was within normal limits, no significant valvular abnormality was identified, and no pericardial effusion was observed.

Gross examination of the fetus showed a female infant of 31 weeks' gestational age weighing 760 g - markedly below the expected weight for gestational age, consistent with severe fetal growth restriction - with grade III maceration and no major external congenital anomaly. Gross placental examination revealed multiple well-demarcated grayish-white to pale-yellow parenchymal areas consistent with placental infarction, without retroplacental hematoma; histopathological examination was not performed. The patient was managed by a multidisciplinary team comprising obstetrics and gynecology, cardiology, general practitioners, nurses, and midwives. Blood pressure remained elevated postpartum, requiring continued antihypertensive therapy, and she was referred to cardiology for ongoing follow-up and evaluation of the ASD.

Discussion

Atrial septal defect (ASD) is a common congenital cardiac lesion that is frequently underdiagnosed until adulthood, often because affected individuals remain free of overt symptoms for decades [1]. In a moderate secundum defect with preserved biventricular function, the resulting right heart volume overload accumulates gradually rather than abruptly, which explains why exertional intolerance can escape clinical attention until an additional hemodynamic stressor, such as pregnancy, is superimposed [2]. Contemporary reviews of pregnancy in moderate-to-complex CHD reinforce that this gradual accumulation of right heart load is precisely why such lesions are so often first unmasked during gestation rather than earlier in life [2,4]. Notably, this patient's presentation reflects not one but two layers of undetected disease: in addition to her unrecognized ASD, her hypertension had already been present before conception yet was never evaluated or treated, consistent with reports that a substantial proportion of women with chronic hypertension enter pregnancy without a prior diagnosis or antihypertensive therapy [14]. This pattern is compounded by the physiological fall in blood pressure during the first half of pregnancy, which can further mask pre-existing chronic hypertension during routine prenatal visits [8]. Adding to both of these gaps, routine antenatal care in Indonesia does not currently incorporate systematic cardiac auscultation or echocardiographic screening [9], a limitation that mirrors current WHO antenatal care standards, which likewise do not mandate cardiac evaluation as part of routine antenatal contacts [12], so that even a soft murmur or a subtly split second heart sound would be unlikely to prompt further cardiac evaluation.

The relationship between maternal ASD and adverse pregnancy outcomes observed in this case is consistent with evidence that congenital heart disease in pregnancy is associated with a significantly increased risk of preeclampsia [5]. Placental factors have similarly been proposed as a mechanistic link between maternal cardiac disease and offspring outcomes, lending plausibility to the placental infarction and fetal growth restriction identified in the present case [6]. This association is further corroborated by a large nationwide Norwegian cohort, which found higher rates of preeclampsia and adverse neonatal outcomes among pregnancies complicated by maternal CHD [7]; however, evidence from a Swedish register-based study suggests this risk is lesion-specific rather than uniform across all cardiac conditions, with valvular disease-but not heart failure broadly-independently associated with increased odds of preterm preeclampsia [15], underscoring that the anatomy and hemodynamic profile of the specific lesion, rather than the mere presence of heart disease, likely drives obstetric risk. Comparable reports have described women with previously uncorrected ASD who remained pregnancy-tolerant until pulmonary hypertension or other complications emerged, underscoring the variable and sometimes delayed clinical trajectory of this lesion [16]. Other multidisciplinary case reports of adult congenital heart disease diagnosed during pregnancy similarly highlight that timely recognition, once suspected, allows for safe obstetric planning and improved maternal outcomes [17].

This case also exposes a structural gap in Indonesia's approach to congenital heart disease control, as current national screening policy remains centered on neonatal pulse oximetry rather than on adult or maternal populations [9]. Even this neonatal-focused screening program has so far been implemented only through feasibility studies and localized pilot initiatives rather than as part of a universal national strategy [10]. This pattern is not unique to Indonesia: pulse oximetry-based screening has been prioritized globally precisely because it is simple and inexpensive, yet it remains, by design, incapable of detecting non-cyanotic structural lesions such as ASD in adults [11]. This leaves a paradox in which adults-the population in which ASD is most frequently first identified-remain entirely outside any structured cardiovascular screening pathway [1,11].

Beyond this systemic screening gap, the present case also illustrates specific clinical red flags that, in retrospect, pointed toward an underlying structural cardiac lesion well before echocardiography was performed. Electrocardiographic markers of right heart volume overload, including right axis deviation and an incomplete right bundle branch block with an rSr' pattern, have been shown to correlate with hemodynamic abnormalities in adults with uncorrected secundum ASD [18]. Because ASD remains a diagnosis that is frequently delayed into adulthood, such electrocardiographic findings warrant particular attention when they co-occur with radiographic cardiomegaly, as both were already present in this patient at admission [19]. A comparable case of postpartum dyspnea and severe hypertension later attributed to previously undiagnosed congenital heart disease similarly illustrates how these signs can be misattributed to more common obstetric or pulmonary causes if a structural cardiac lesion is not specifically considered [20]. In the present case, these radiographic and electrocardiographic clues were documented on admission but were acted upon only after intrauterine fetal death had already occurred, underscoring a missed opportunity for earlier cardiology involvement - a gap with consequences that extend beyond this single case, given that stillbirth from preventable cause, including undetected maternal cardiovascular disease, remains a persistent and under-addressed burden in low- and middle-income countries [13].

We therefore propose that antenatal care standards incorporate basic cardiac screening, including careful auscultation and a low threshold for electrocardiographic or echocardiographic referral, particularly when hypertension proves difficult to control or when compatible radiographic or electrocardiographic signs are present. International scientific statements now recommend that pregnant women with suspected or confirmed cardiovascular disease be managed within a multidisciplinary cardio-obstetric or pregnancy heart team as early as possible in pregnancy [4,21]. This recommendation is reinforced by cohort data showing that most major cardiovascular events during pregnancy occur in women whose cardiovascular disease was unknown before conception, precisely the scenario illustrated by the present case [22]. Establishing such a pathway in Indonesian antenatal care could allow congenital heart disease to be identified and optimized before, rather than after, a catastrophic obstetric outcome occurs [3].

Conclusion

This case shows that an atrial septal defect - compounded by pre-existing, unrecognized chronic hypertension - can remain silent throughout pregnancy and surface only after a catastrophic complication such as intrauterine fetal death from preeclampsia. It underscores the need to extend Indonesia's congenital heart disease screening beyond the neonatal period to reproductive-age women, and to recognize refractory hypertension with radiographic or electrocardiographic signs of right heart overload as a clue warranting cardiology evaluation, not preeclampsia alone. A multidisciplinary cardio-obstetric approach could enable earlier recognition and better maternal and fetal outcomes in future pregnancies. A silent heart is not a healthy one - in obstetric care, that silence should never be measured in stillbirths.

Authorship Criteria and Contributions

KAHK, EDY, FPA, and ERMP contributed equally to this work as co-authors, jointly involved in patient data collection, and manuscript drafting. KAHK managed the patient's obstetric care and led the drafting of the manuscript. EDY performed and interpreted the echocardiographic evaluation and contributed to the discussion section. FPA contributed to case supervision and manuscript revision. ERMP contributed to case supervision and is the guarantor of this case report. KAHK and EDY were responsible for the final compilation and finalization of the manuscript. TARK, as the supervising obstetrician, provided critical revision and minor corrections to the final manuscript prior to submission.

Conflict of Interest

The authors declare no conflicts of interest related to this publication.

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