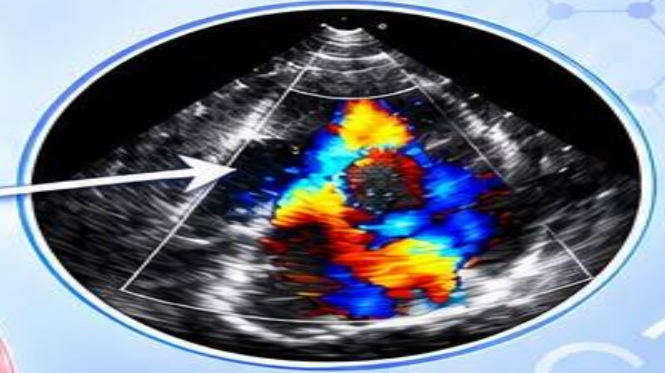
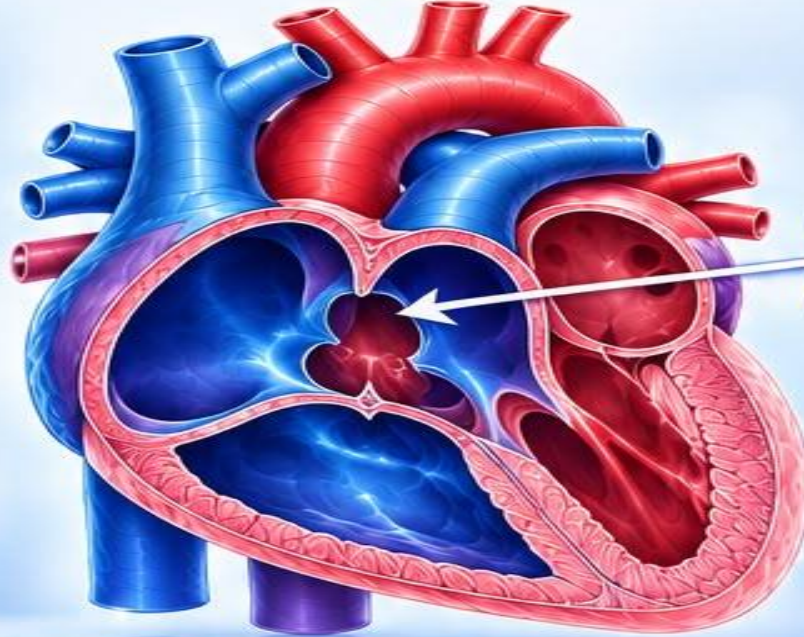


Pregnancy and Congenital Heart Disease :

A Case of Atrial Septal Defect (Sinus Venosus Type)



SPEAKER

Dr. Md. Ali Ehsan Siddiqui

*Professor and Head
Department of Cardiology*

Asymptomatic Sinus Venosus ASD in a Primigravida

Unexpected diagnosis during routine antenatal screening in a previously healthy young woman

PATIENT PROFILE & INCIDENTAL FINDING

- **Patient:** A 26-year-old woman in her first pregnancy (primigravida).
- **Prior Health:** Considered herself completely healthy from childhood with no known chronic conditions.
- **Incidental Detection:** Sinus Venosus type Atrial Septal Defect (ASD) detected during routine antenatal echocardiography.
- **Silent Pathophysiology:** No antecedent heart symptom or cardiovascular evaluation performed prior to this pregnancy.

HEMODYNAMIC RESILIENCE IN PREGNANCY

- **Preserved Function:** Despite significant congenital heart disease, she maintained excellent functional capacity throughout pregnancy.
- **Right Heart Loading:** Tolerated marked gestational blood volume expansion and progressive right-sided volume loading.
- **Elevated Pressures:** Demonstrated substantial pulmonary pressure elevation (PASP 56 mmHg) without acute decompensation.
- **Clinical Course:** Remained stable under medical therapy, allowing gestation to progress to term safely.

KEY CLINICAL TAKEAWAY

Pregnancy acts as a physiological "stress test" on the cardiovascular system. In acyanotic congenital heart defects like ASD, increased circulatory volume can unmask occult lesions while preserved ventricular compliance allows successful maternal-fetal outcomes under vigilant multidisciplinary care.

Patient Demographics & Obstetric Timeline

Baseline characteristics and initial admission echocardiographic alert

PATIENT PROFILE

- **Age:** 26-year-old female.
- **Obstetric Index:** Primigravida (Gravida 1, Para 0).
- **Marital History:** Married for 1 year; harmonious family life.
- **Development:** Mentally and physically normal from childhood.
- **Menstrual History:** Normal, regular menstrual cycles prior to conception.

OBSTETRIC TIMELINE

- **LMP:** 05.12.2025 (Last Menstrual Period).
- **EDD:** 11.09.2026 (Estimated Date of Delivery).
- **Admission Date:** 13.09.2026 (~40+ weeks of gestation).
- **Prior Diagnosis:** No previous diagnosis of congenital heart disease (CHD).
- **Detection Mode:** ASD detected incidentally on routine antenatal echocardiography.

INITIAL ECHO SCREENING ALERT

- **Lesion:** ASD Sinus Venosus type with left-to-right interatrial shunt.
- **Chambers:** Significant Right Atrial (RA) and Right Ventricular (RV) dilatation.
- **Tricuspid Valve:** Moderate tricuspid regurgitation (TR).
- **Pulmonary Pressure:** Elevated PASP documented on Doppler.
- **LV Function:** Preserved Left Ventricular Systolic Function (LVEF 66%).

Clinical Latency: Absence of Overt Heart Failure

How a significant intracardiac shunt remained asymptomatic prior to pregnancy

CARDIAC SYMPTOM HISTORY

- **Chest Discomfort:** No history of angina, chest tightness, or heaviness.
- **Syncope / Presyncope:** No episodes of fainting, lightheadedness, or blackouts.
- **Orthopnea / PND:** No nocturnal cough or posture-related breathlessness.

CYANOSIS & OXYGENATION

- **Cyanosis:** Completely absent throughout childhood and pregnancy.
- **Oxygen Saturation:** Maintained normal SpO2 (>98%) on ambient air.
- **Clubbing:** No digital clubbing or peripheral stigmata of chronic hypoxemia.

RHYTHM & PALPITATION HISTORY

- **Palpitations:** No history of rapid, fluttering, or pounding heartbeats.
- **Atrial Arrhythmias:** No documented supraventricular tachycardia, AF, or flutter.
- **Clinical Relevance:** Atrial stretch often triggers arrhythmias; patient remained in sinus rhythm.

RESPIRATORY & FUNCTIONAL STATUS

- **Respiratory Infections:** No history of recurrent childhood lower respiratory tract infections.
- **Functional Capacity:** Normal functional capacity prior to pregnancy.
- **Exercise Tolerance:** Able to perform daily household activities without limitation.

Summary: Clinical latency is classic in adult ASD where low left-to-right pressure gradient delays overt congestive symptoms.

Antenatal Medical Regimen & Team Consultation

Targeted pharmacotherapy for gestational hypertension and pulmonary vascular modulation

GESTATIONAL HYPERTENSION

- **Indication:** Developed gestational hypertension during late antenatal course.
- **Oral Labetalol:** Combined alpha/beta-adrenergic blocker; safe, effective first-line agent in pregnancy.
- **Oral Methyldopa:** Centrally acting alpha-2 agonist; added for synergistic blood pressure control.
- **Goal:** Prevent hypertensive crises and reduce LV afterload without impairing uteroplacental blood flow.

RIGHT HEART & PULMONARY UNLOADING

- **Oral Furosemide:** Judicious low-dose loop diuretic to relieve right ventricular volume overload.
- **Fluid Balance:** Carefully titrated to prevent peripheral edema while avoiding maternal hypovolemia.
- **Oral Sildenafil:** Phosphodiesterase-5 (PDE-5) inhibitor; prescribed for pulmonary arterial hypertension.
- **Mechanism:** Promotes pulmonary vasodilation, reducing pulmonary vascular resistance and RV afterload.

CARDIO-OBSTETRIC CONSULTATION

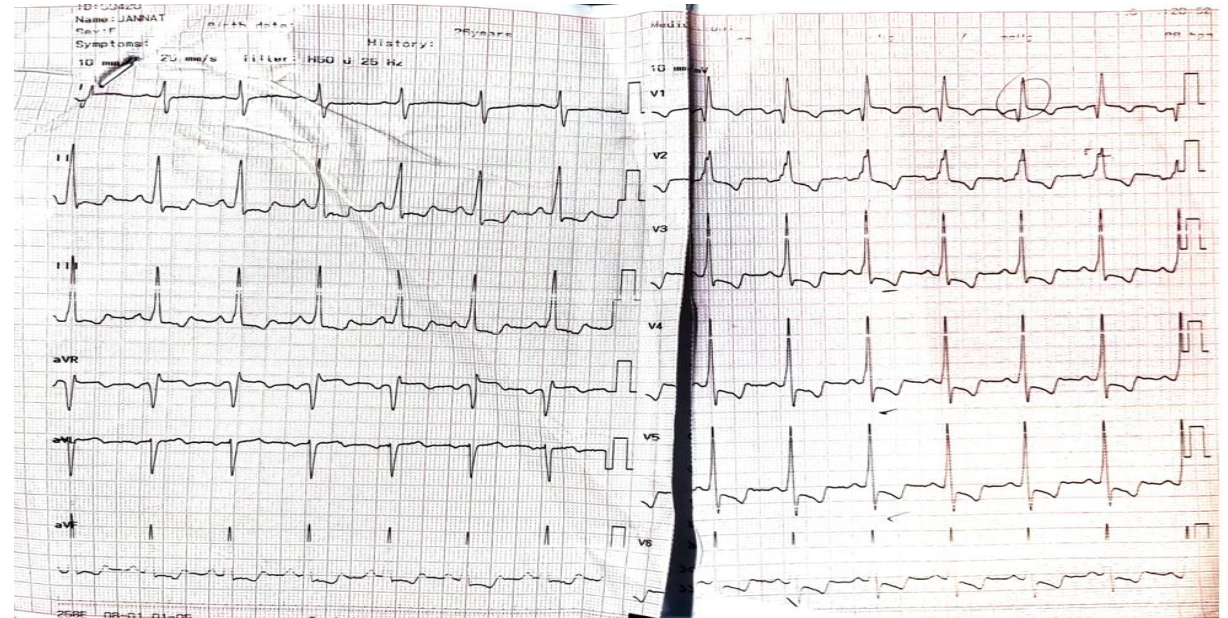
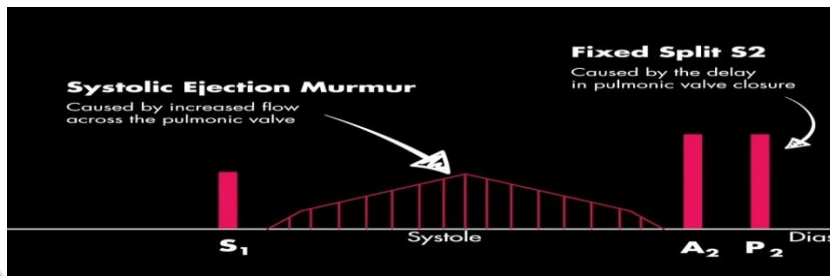
- **Cardiology Input:** Formal cardiology consultation obtained following echocardiographic alert.
- **Multidisciplinary Plan:** Joint consensus between Cardiology, Obstetrics, and Anesthesia.
- **Delivery Strategy:** Advised planned normal delivery with strict intrapartum and hemodynamic care.
- **Postpartum Plan:** Transfer to Coronary Care Unit (CCU) planned for post-delivery period.

Bedside Auscultation & 12-Lead Electrocardiogram

Auscultatory hallmarks of ASD and characteristic right ventricular conduction changes

BEDSIDE PHYSICAL EXAMINATION

- **Loud P2:** Accentuated pulmonic component of second sound auscultated at 2nd left intercostal space; reflects elevated pulmonary artery systolic pressure (PASP 56 mmHg).
- **Systolic Flow Murmur:** Grade 2/6 midsystolic ejection murmur over the pulmonary area; caused by massive hyperdynamic stroke volume traversing a structurally normal pulmonary valve.
- **Fixed Wide Splitting of S2:** Pathognomonic hallmark of hemodynamically significant ASD; Fixed A2–P2 splitting due to reciprocal respiratory shifts in systemic venous return and interatrial shunt volume.



ECG Interpretation: Sinus rhythm (HR ~78 bpm), Right Axis Deviation (+110°), Incomplete RBBB pattern (rSR' in lead V1 - circled) reflecting RV volume overload, and anterior T-wave inversion (V1–V3) reflecting RV strain.

Transthoracic Echocardiogram: 2D Structural Findings

Chamber morphology, septal defect visualization, and M-mode chamber dimensions

2D MORPHOLOGY & SEGMENTAL ANATOMY

- **Cardiac Position:** Situs solitus, levocardia, concordant AV & ventriculoarterial connections; normally related great arteries.
- **Interatrial Septum (IAS):** Echo dropout identified in the superior aspect of the IAS near the SVC junction, confirming Sinus Venosus ASD.
- **Interventricular Septum (IVS):** Intact septum. Displays marked flattening resulting in a D-shaped LV in both systole and diastole (sign of combined RV volume & pressure overload).
- **Chamber Sizes:** Marked Right Atrial (RA) and Right Ventricular (RV) dilatation. Left Atrium within normal limits.
- **Valvular Morphology:** MV, PV, AV, and TV structurally normal in morphology and leaflet motion.
- **Pericardium & Masses:** No pericardial effusion; no intracardiac thrombus or vegetation.

Parameter	Measured Value	Status / Ref.
Aortic Root (AO)	22 mm	Normal (20–37 mm)
Aortic Cusp Sep (ACS)	20 mm	Normal (15–26 mm)
Left Atrium (LA)	37 mm	Normal (<40 mm)
IVS Diastole (IVSd)	08 mm	Normal (6–11 mm)
LV Post Wall (LVPWd)	08 mm	Normal (6–11 mm)
LV Diastolic (LVDd)	32 mm	Normal (35–56 mm)
LV Systolic (LVDs)	20 mm	Normal (20–40 mm)
Fractional Shortening (FS)	37%	Normal (25–45%)
LV Ejection Fraction (EF)	68%	Preserved (>55%)
TAPSE (RV Function)	17 mm	Preserved (>16 mm)

Doppler Echocardiography: Valvular Hemodynamics

Transvalvular flow velocities, pressure gradients, and regurgitation assessment

VALVULAR DOPPLER STUDY PARAMETERS

Valvular Orifice	Flow Velocity (m/sec)	Peak Pressure Gradient (PPG)	Regurgitation Grade
Mitral Valve (MV)	0.75 m/sec	2.26 mmHg	Nil (Competent)
Aortic Valve (AV)	0.88 m/sec	3.11 mmHg	Nil (Competent)
Pulmonary Valve (PV)	1.17 m/sec	5.51 mmHg	Moderate PR (Hyperdynamic flow)
Tricuspid Valve (TV)	0.53 m/sec	1.12 mmHg	Moderate TR (Used for PASP calculation)
MR dp/dt	—	mmHg/s	Nil

FLOW GRADIENT INTERPRETATION

- **Pulmonary Flow:** Peak pulmonary flow velocity of 1.17 m/s (PPG 5.51 mmHg) reflects high-volume transpulmonary stroke volume rather than valvular stenosis.
- **Left-Sided Valves:** Mitral and aortic velocities are entirely normal, confirming absence of left-sided obstruction.

COLOR FLOW & SHUNT SUMMARY

- **Color Flow Doppler:** Confirmed left-to-right interatrial shunting across upper interatrial septum; no abnormal jet across IVS.
- **Tricuspid Regurgitation:** Moderate TR jet allowed continuous-wave Doppler measurement of peak TR velocity, deriving PASP of 56 mmHg.

Final Echocardiographic Impression

Integrated synthesis of anatomical defects, chamber remodeling, and hemodynamics

01 | CONGENITAL DEFECT — Sinus Venosus ASD

Echo dropout visualized in upper part of IAS near SVC junction, with continuous left-to-right shunting.

02 | CHAMBER REMODELING — Dilated RA & Dilated RV

Prominent enlargement of right-sided chambers secondary to chronic volume overload; D-shaped LV in systole & diastole.

03 | LV SYSTOLIC FUNCTION — Good LV Systolic Function (68%)

LVEF calculated at 68% (FS 37%); no regional wall motion abnormalities (RWMA); normal wall thickness.

04 | RV SYSTOLIC FUNCTION — Preserved RV Systolic Function

TAPSE measured at 17 mm (normal >16 mm), confirming preserved longitudinal right ventricular contractility.

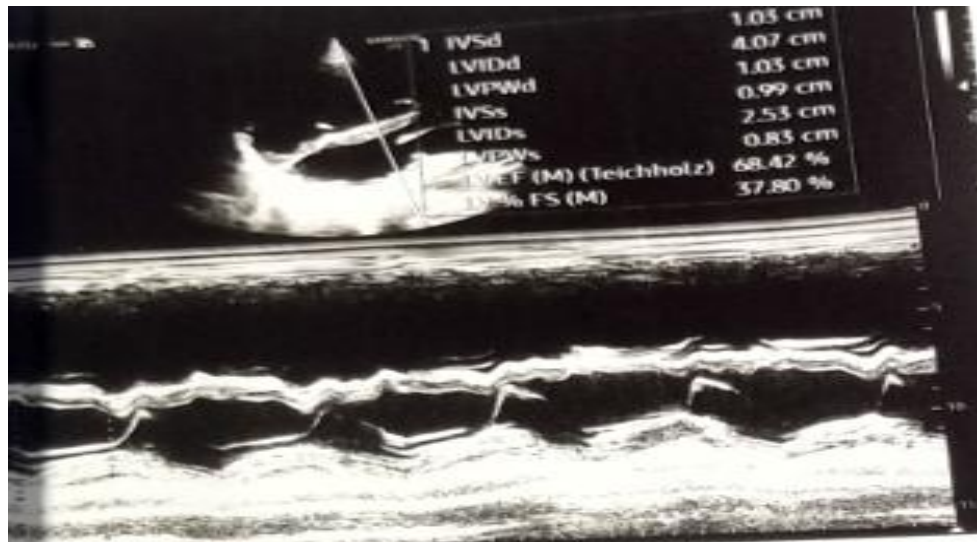
05 | PULMONARY HEMODYNAMICS — PASP (56 mmHg)

Doppler-derived PASP of 56 mmHg; moderate-to-severe pulmonary hypertension driven by hyperkinetic pulmonary flow.

Echocardiographic Modalities: M-Mode & Color Doppler

Visual confirmation of left ventricular mechanics and tricuspid regurgitant jet velocity

PARASTERNAL M-MODE ECHOCARDIOGRAM



- M-Mode LV Chamber Dimensions & Teichholz EF:
- IVSd: 1.03 cm | LVIDd: 4.07 cm | LVPWd: 1.03 cm
 - IVSs: 0.99 cm | LVIDs: 2.53 cm | LVPWs: 0.83 cm
 - Teichholz LVEF: 68.42% | Fractional Shortening: 37.80%

COLOR DOPPLER TRICUSPID REGURGITANT JET



- TR Jet Interrogation & Pulmonary Artery Pressure:
- Continuous wave Doppler interrogation of peak TR jet
 - TV Vmax / TV PGmax used to calculate $PASP = 4(V_{max})^2 + RAP$
 - Yields estimated PASP of 56 mmHg (Moderate-Severe PH)

Coronary Care Unit (CCU) Management

Close hemodynamic surveillance during the critical post-delivery autotransfusion window

PATIENT IN CCU (POSTPARTUM DAY 1)



CCU ADMISSION PROTOCOL (BED CCU-08)

- **Immediate Transfer:** Transferred directly from labor room to CCU for continuous hemodynamic observation.
- **Critical Rationale:** Postpartum hemodynamic volume shift / autotransfusion of blood creates peak danger window for acute RV volume overload.

MONITORING & SUPPORTIVE CARE

- **Cardiac Monitoring:** Continuous ECG rhythm tracking to detect arrhythmia (atrial flutter or supraventricular tachycardias).
- **Respiratory Support:** Maintained on low-flow supplemental oxygen via nasal cannula; continuous pulse oximetry monitoring.

CLINICAL OUTCOME & MATERNAL STABILITY

- **Clinical Status:** Patient remained completely awake, alert, seated upright, smiling, and comfortable.
- **No Heart Failure:** No peripheral edema, hepatomegaly, pulmonary crepitations, or signs of acute right-heart failure.

Successful Delivery & Maternal-Neonatal Outcome

Smooth labor course, healthy neonate, and safe hospital discharge

THE BIRTH EVENT

- **Date & Time:** 13.09.2026 at 12:20 PM.
- **Mode of Delivery:** Spontaneous normal delivery with normal intrapartum course.
- **Neonatal Outcome:** Delivered a healthy, alive female baby.
Normal cry at birth with normal Apgar scores.
- **No Intrapartum Crises:** Zero hemodynamic collapse, sustained arrhythmias, or sudden desaturation during labor.

POSTPARTUM CCU SURVEILLANCE

- **Observation Window:** Monitored closely in CCU for 24 hours.
- **Oxygen Therapy:** Low-flow O2 therapy maintained to reduce pulmonary vascular resistance.
- **Fluid Restrictions:** Strict fluid charting to prevent iatrogenic volume overload.

HOSPITAL DISCHARGE & FOLLOW-UP

- **Discharge Date:** Discharged home in stable clinical condition on 17.09.2026.
- **Total Stay:** 4 days total hospital stay following delivery.
- **Postpartum Advice:** Advised ongoing medical compliance, infant care support.
- **Definitive Plan:** Outpatient cardiology follow-up scheduled for elective surgical ASD closure evaluation.

Pregnancy and Atrial Septal Defect

Hemodynamic interactions, risk determinants, and evidence-based cardio-obstetric management

UNCOMPLICATED ASD

- Pregnancy is generally well tolerated in uncomplicated cases.
- Normal functional capacity often maintained throughout gestation.

SHUNT AUGMENTATION

- Gestational blood volume expansion increases left-to-right shunt.
- Right ventricular volume loading accelerates chamber dilatation.

DETERMINANTS OF RISK

- RV volume overload magnitude.
- Pulmonary artery systolic pressure.
- Right and left ventricular function.
- Underlying symptom status (NYHA).

Adult Congenital Heart Disease: The LMIC Context

Challenges of late presentation and unmasking during pregnancy in Bangladesh

THE SILENT BURDEN IN DEVELOPING NATIONS

- **Undiagnosed Survival:** In Bangladesh and similar low-and-middle-income countries (LMICs), many adults with CHD reach reproductive age completely undiagnosed.
- **Clinically Silent Lesions:** Acyanotic shunts like ASD cause little cyanosis or distress in childhood, frequently escaping detection in the absence of routine school or neonatal screening.
- **Healthcare Access Gaps:** Late presentations are common due to limited access to routine pediatric cardiology evaluation in rural and semi-urban populations.

PREGNANCY AS A DIAGNOSTIC OCCASION

- **First Medical Contact:** Pregnancy is frequently the very first time a young woman undergoes formal medical examination and cardiovascular auscultation.
- **Hemodynamic Unmasking:** The ~50% increase in blood volume during pregnancy places an unprecedented workload on the heart, making murmurs and chamber dilatation clinically noticeable.
- **Diagnostic Role of Echo:** Antenatal echocardiography often serves as the initial diagnostic occasion that saves lives through timely multidisciplinary planning.

PUBLIC HEALTH IMPERATIVE

Maternal cardiovascular disease is now a leading non-obstetric cause of maternal mortality. Early antenatal cardiovascular assessment and universal access to basic echocardiographic screening are vital steps to curb preventable maternal deaths in developing countries.

Atrial Septal Defect: Definition & Anatomical Variants

Abnormal communication between atria resulting in shunting of blood

1. OSTIUM SECUNDUM ASD (~75%)

- **Location:** Located in the region of the fossa ovalis (mid-septum).
- **Frequency:** Most common variant of all ASDs (approx. 75%).
- **Management:** Ideal candidate for percutaneous transcatheter device closure (e.g. Amplatzer) if adequate tissue rims exist.

2. OSTIUM PRIMUM ASD (~15–20%)

- **Location:** Situated low in the interatrial septum, adjacent to the atrioventricular valves.
- **Spectrum:** Part of the endocardial cushion defect / atrioventricular septal defect (AVSD) spectrum.
- **Features:** Frequently associated with a cleft mitral valve; requires surgical patch repair.

3. SINUS VENOSUS ASD (~5–10%) — THIS PATIENT

- **Location:** Usually located near the SVC–RA junction; less commonly near IVC entry.
- **PAPVC:** Strongly associated with Partial Anomalous Pulmonary Venous Connection (PAPVC).
- **Management:** Not suitable for transcatheter devices; requires open surgical patch baffling.

4. CORONARY SINUS DEFECT (<1%)

- **Location:** Characterized by partial or total unroofing of the coronary sinus wall into the left atrium.
- **Frequency:** Rarest anatomical form of ASD (<1%).
- **Features:** Often associated with a persistent left superior vena cava (PLSVC); requires surgical reconstruction.

Hemodynamic Significance of Interatrial Shunting

The pathophysiological cascade from volume overload to pulmonary vascular remodeling

STAGE 1: SHUNT DYNAMICS

- **Pressure Gradient:** Left atrial pressure normally exceeds right atrial pressure by 2–4 mmHg.
- **Compliance Difference:** The right ventricle is significantly more compliant and thinner than the left ventricle.
- **Left-to-Right Shunting:** Blood preferentially shunts from LA to RA across the defect throughout both systole and diastole.
- **Determinants:** Shunt volume depends on defect size, chamber compliances, and vascular resistance ratios.

STAGE 2: CHAMBER OVERLOAD

- **RA & RV Dilatation:** Chronic volume overload leads to progressive dilatation of the right atrium and right ventricle.
- **Pulmonary Hypercirculation:** Pulmonary blood flow (Q_p) exceeds systemic blood flow (Q_s), often reaching $Q_p:Q_s > 2:1$.
- **Septal Flattening:** RV volume expansion flattens the interventricular septum, generating the classic D-shaped LV on echo.
- **Conduction Delay:** Right ventricular stretching causes crista supraventricularis hypertrophy and incomplete RBBB.

STAGE 3: PULMONARY VASCULAR IMPACT

- **Endothelial Shear Stress:** Decades of hyperkinetic pulmonary circulation induce endothelial injury and medial hypertrophy.
- **Pulmonary Hypertension:** Pulmonary vascular resistance (PVR) progressively rises, elevating pulmonary artery systolic pressure (PASP).
- **Longstanding Untreated:** Severe untreated cases can progress to irreversible vascular remodeling.
- **Eisenmenger Physiology:** If PVR exceeds SVR, shunt reversal (right-to-left) occurs, causing cyanosis and high maternal mortality.

Cardiovascular Adaptations in Pregnancy & ASD Interaction

How physiological gestational circulatory expansion augments interatrial shunting

PLASMA VOLUME

+40% to +50%

Peak expansion reached by 32–34 weeks; increases systemic venous return.

CARDIAC OUTPUT

+30% to +50%

Rises early from 5th week; mediated by stroke volume & maternal heart rate.

HEART RATE

+15 to +20 bpm

Baseline resting tachycardia increases total daily cardiac cycles.

STROKE VOLUME

+30%

Increases primarily in first and second trimesters due to increased preload.

PATHOPHYSIOLOGICAL INTERACTION WITH ATRIAL SEPTAL DEFECT

- **Augmented Shunt Fraction:** Massive expansion in intravascular volume elevates systemic venous return and left atrial filling, significantly increasing the left-to-right shunt volume across the ASD.
- **Exaggerated RV Loading:** The already dilated right ventricle is forced to accommodate both gestational hypervolemia and augmented interatrial shunt volume, accelerating RV volume stress.
- **Unmasking Latent Pathology:** These combined hemodynamic demands explain why previously asymptomatic, healthy young women frequently present for the first time during pregnancy with cardiac murmurs, dyspnea, or elevated pulmonary pressures.

Maternal Risk Stratification in Pregnancy with ASD

Differentiating favorable uncomplicated cases from high-risk clinical profiles

FAVORABLE / UNCOMPLICATED ASD (mWHO II)

- **General Prognosis:** Most women with an uncomplicated ASD tolerate pregnancy remarkably well.
- **Defect Size:** Small to moderate defect with preserved ventricular compliance.
- **Pulmonary Pressures:** Normal or only mildly elevated pulmonary artery pressures.
- **Ventricular Function:** Normal or mildly enlarged RV with preserved systolic function (TAPSE >16 mm).
- **Symptom Status:** NYHA Functional Class I or II; excellent functional capacity.
- **Outcome:** Favorable maternal and neonatal outcomes; planned normal delivery appropriate.

HIGH-RISK FEATURES (mWHO III / IV)

- **Severe RV Dilatation:** Significant RV volume overload with right ventricular systolic dysfunction.
- **Pulmonary Hypertension:** Severe pulmonary arterial hypertension (PAH) with elevated pulmonary vascular resistance.
- **Atrial Arrhythmias:** Supraventricular tachycardias, atrial flutter, or atrial fibrillation due to chronic atrial stretch.
- **Heart Failure:** Overt right-sided congestive failure, hepatomegaly, or systemic venous congestion.
- **Cyanosis / Eisenmenger:** Reversal of interatrial shunt (right-to-left) or Eisenmenger syndrome (mWHO Class IV - maternal mortality 20–50%).

Sinus Venosus ASD: Anatomy & Surgical Considerations

Distinct anatomical relationships, anomalous venous drainage, and repair strategy

INCIDENCE & ANATOMY

- **Incidence:** Accounts for approximately 5–10% of all atrial septal defects.
- **SVC Type:** Most commonly located superiorly near the junction of the Superior Vena Cava (SVC) and Right Atrium (RA).
- **IVC Type:** Less commonly located inferiorly near the IVC orifice (<2% of cases).
- **Embryology:** Results from deficiency of the common wall between the superior vena cava and the right upper pulmonary vein.

ASSOCIATION WITH PAPVC

- **High Coincidence:** Frequently (>80–90%) associated with Partial Anomalous Pulmonary Venous Connection (PAPVC).
- **Venous Drainage:** Right pulmonary veins drain anomalously into the SVC or directly into the high RA.
- **Shunt Magnification:** Anomalous pulmonary veins directly augment right atrial volume loading, intensifying the left-to-right shunt fraction.

SURGICAL REPAIR STRATEGY

- **No Device Closure:** Unsuitable for percutaneous transcatheter device closure due to complete absence of superior septal rim.
- **Open Surgical Repair:** Mandates open surgical baffling using single-patch, double-patch, or the Warden procedure.
- **Warden Procedure:** Transects SVC above anomalous veins, anastomosing SVC to RAA while baffling anomalous veins through ASD into LA.
- **Timing:** Electively scheduled postpartum after pregnancy hemodynamics normalize.

Interpreting Elevated PASP in Pregnancy: 56 mmHg

Differentiating hyperkinetic pulmonary flow from fixed pulmonary arterial hypertension

KEY DIAGNOSTIC POINTS: DOPPLER PASP

- **Doppler Measurement:** Continuous-wave Doppler TR jet velocity derived an estimated PASP of 56 mmHg, raising immediate concern for pulmonary hypertension.
- **PASP vs True PAH:** Elevated Doppler PASP alone does NOT establish irreversible pulmonary arterial hypertension (PAH) or fixed vascular disease.
- **Hyperkinetic Flow Effect:** In pregnancy, the 50% expansion in cardiac output combined with a large left-to-right shunt creates massive pulmonary blood flow.
- **Pressure Equation:** $\text{Pressure} = \text{Flow} \times \text{Resistance}$. Even with normal or minimally elevated PVR, massive transpulmonary flow generates high systolic pressures.

WHY CONSERVATIVE MANAGEMENT SUCCEEDED

- **Preserved RV Function:** TAPSE was preserved at 17 mm, proving the right ventricle was compliant, contractile, and not failing.
- **Absent Cyanosis:** Absence of cyanosis or right-to-left shunting confirmed that systemic vascular resistance remained higher than pulmonary resistance.
- **Good Functional Class:** Patient maintained NYHA Class I–II without rest dyspnea or signs of low cardiac output.
- **Targeted Pharmacotherapy:** Sildenafil reduced pulmonary vascular tone while Furosemide optimized preload, allowing safe continuation to full-term delivery.

Multidisciplinary Cardio-Obstetric Management Principles

Evidence-based framework for comprehensive maternal and fetal surveillance

1. MULTIDISCIPLINARY TEAM

Integrated cardio-obstetric team including Adult Congenital Heart Disease (ACHD) cardiologists, maternal-fetal medicine specialists, obstetric anesthesiologists, and neonatologists.

2. CLINICAL SURVEILLANCE

Frequent clinical review assessing for exertional dyspnea, orthopnea, peripheral edema, palpitations, presyncope, and monitoring arterial oxygen saturation.

3. SERIAL ECHOCARDIOGRAPHY

Echocardiographic surveillance performed each trimester and peripartum to monitor RV dimensions, TAPSE, septal dynamics, and pulmonary artery systolic pressures.

4. FLUID STEWARDSHIP

Strict avoidance of both excessive iatrogenic volume loading (which precipitates RV failure) and severe hypovolemia (which impairs cardiac output).

5. ARRHYTHMIA & FAILURE CARE

Prompt diagnosis and treatment of atrial tachyarrhythmias (beta-blockers, electrical cardioversion); judicious use of diuretics for right heart congestion.

6. PLANNED DEFECT CLOSURE

Definitive ASD repair electively planned either prior to conception or 3–6 months postpartum, following physiological recovery.

Mode of Delivery: Normal Delivery vs Cesarean Section

Cardiology consensus on optimal mode of delivery in adult congenital heart disease

CORE CARDIO-OBSTETRIC PRINCIPLE

“Most women with an uncomplicated Atrial Septal Defect (ASD) can have a normal delivery. ASD itself is NOT an indication for Cesarean Section.”

PLANNED NORMAL DELIVERY CRITERIA

- **Defect Status:** Uncomplicated small, moderate, or hemodynamically compensated large ASD.
- **Pulmonary Pressure:** Absence of severe pulmonary arterial hypertension or fixed vascular resistance.
- **Shunt Direction:** Absence of cyanosis or right-to-left shunt reversal.
- **Ventricular Function:** Normal or mildly enlarged RV with preserved systolic function (TAPSE > 16 mm).
- **Rhythm & Failure:** No sustained, unmanageable arrhythmias or refractory heart failure.

CESAREAN SECTION INDICATIONS IN ASD

- **Obstetric Indications:** Cesarean delivery is primarily reserved for standard obstetric indications (e.g. cephalopelvic disproportion, placenta previa, malpresentation, fetal distress).
- **Cardiac Exceptions:** Rare cardiac indications include refractory severe heart failure, severe unstable Eisenmenger syndrome, or decompensated aortic pathology.
- **Guideline Stance:** ESC and AHA/ACC guidelines emphasize that unnecessary cesarean sections increase surgical morbidity without cardiovascular benefit.

Why Normal Delivery is Preferred in ASD Patients

Hemodynamic, surgical, and thrombotic advantages over cesarean section

1. LOWER BLOOD LOSS

- **Volume Difference:** Normal delivery typically involves 300–500 mL of blood loss, compared to 800–1000 mL or more during cesarean section.
- **Preload Stability:** Minimizes sudden, precipitous drops in systemic venous return that compromise cardiac output.

2. LESS ABRUPT HEMODYNAMIC STRESS

- **Gradual Shifts:** Uterine contractions produce rhythmic, predictable circulatory changes rather than sudden surgical volume shifts.
- **Physiological Buffering:** Preserves natural compensatory vascular adjustments in stable ASD patients.

3. AVOIDS SURGICAL & ANESTHETIC RISKS

- **Anesthesia Hazards:** Avoids myocardial depression, positive-pressure ventilation risks of general anesthesia, and profound spinal vasodilation.
- **Infection & Wound Risk:** Eliminates major abdominal surgery, wound infection, and peritoneal inflammation.

4. REDUCED THROMBOEMBOLIC RISK

- **Immediate Ambulation:** Vaginal delivery facilitates rapid postpartum mobilization, drastically cutting deep vein thrombosis (DVT) risk.
- **Paradoxical Embolism:** In ASD, any DVT carries the catastrophic risk of crossing the defect into systemic arterial circulation (stroke / paradoxical embolus).

Intrapartum Management & Monitoring During Labor

Targeted hemodynamic safeguards, fluid protocols, and analgesia in labor

HEMODYNAMIC & SPO2 SURVEILLANCE

- **Continuous SpO2:** Continuous pulse oximetry monitoring to immediately catch systemic desaturation or shunt reversal.
- **Cardiac Monitoring:** Continuous ECG rhythm tracking to detect atrial tachyarrhythmias triggered by labor catechols.
- **Blood Pressure:** Frequent non-invasive (or arterial line if unstable) blood pressure surveillance.
- **Oxygen Support:** Administer supplemental nasal oxygen to maintain optimal alveolar oxygenation and reduce PVR.

FLUID STEWARDSHIP & RESTRICTIONS

- **Avoid Overload:** Strictly restrict intravenous fluid maintenance; avoid rapid fluid boluses that acutely overload the RV.
- **Fluid Balance Chart:** Maintain hourly intake-and-output charting throughout labor and immediate postpartum.
- **Diuretic Access:** Intravenous furosemide readily accessible at bedside if acute pulmonary venous or right-heart congestion develops.

ANALGESIA & LABOR FACILITATION

- **Epidural Analgesia:** Carefully titrated, slow-onset epidural analgesia effectively blunts pain-induced sympathetic surges and maternal tachycardia.
- **Left Lateral Position:** Deliver in left lateral decubitus tilt to avoid aortocaval compression by the gravid uterus.
- **Second Stage:** Passive fetal descent and assisted second stage (vacuum or forceps) considered to limit strenuous Valsalva pushing.

Important Issue: Pulmonary Hypertension in Labor

How pulmonary hypertension fundamentally elevates risk and transforms peripartum management

CRITICAL RISK FACTOR: PULMONARY HYPERTENSION

“Pulmonary hypertension changes maternal risk substantially. Presence of right-heart dysfunction, cyanosis, or Eisenmenger physiology mandates a multidisciplinary high-risk cardiac/obstetric delivery protocol.”

THE PATHOPHYSIOLOGICAL DANGER

- **Vascular Resistance:** In fixed pulmonary arterial hypertension, pulmonary vessels cannot dilate to accommodate increased cardiac output.
- **Systemic Hypotension Hazard:** If systemic vascular resistance drops (due to vasodilation, anesthesia, or hemorrhage), SVR falls below PVR.
- **Acute Shunt Reversal:** Triggers immediate right-to-left shunting across the ASD, causing profound arterial hypoxemia, acute RV ischemia, and cardiovascular collapse.

HIGH-RISK CARDIAC/OBSTETRIC PLAN

- **Tertiary Environment:** Delivery must occur in a tertiary referral center with adult cardiac intensive care capabilities.
- **Invasive Monitoring:** Arterial line placement for continuous beat-to-beat systemic blood pressure monitoring.
- **Inotropes & Vasodilators:** Targeted pulmonary vasodilators (Inhaled Nitric Oxide, Sildenafil) and vasopressors (Norepinephrine) at bedside.
- **Delivery Route:** Normal delivery may still be possible with meticulous hemodynamic stability, but threshold for intervention is low.

Postpartum Hemodynamics & Definitive ASD Repair

The peripartum autotransfusion window and roadmap to definitive corrective surgery

THE AUTOTRANSFUSION WINDOW

- **Immediate Shift:** Delivery and placental expulsion relieve caval compression, while sustained uterine contraction autotransfuses 500–1000 mL blood into central circulation.
- **Critical 24–72 Hours:** The first 24–72 hours postpartum represent the highest risk window for acute RV volume overload and decompensation.
- **Vigilance:** Mandates strict CCU surveillance, avoiding overhydration, and maintaining oxygenation.

INPATIENT & PUERPERAL CARE

- **CCU:** Step-down care with continuous monitoring, tracking fluid intake-output, and maintaining SBP control.
- **Embolic Protection:** Use intravenous air-filters on all IV lines; vigorous DVT prophylaxis with compression stockings and early ambulation.
- **Safe Discharge:** Discharge achieved safely once hemodynamic stability and functional independence are fully documented.

DEFINITIVE SURGICAL REPAIR

- **Timeline:** Elective surgical correction planned at 3–6 months postpartum.
- **Rationale:** Allows full involution of gestational plasma expansion, cardiac output, and pulmonary pressures to true non-pregnant baseline.
- **Procedure:** Surgical baffling of Sinus Venosus ASD with redirection of anomalous pulmonary veins into left atrium.
- **Prognosis:** Curative intervention offering normal life expectancy and excellent long-term quality of life.

WORLD HEART DAY 2026

"DON'T MISS A BEAT"

THE GLOBAL SILENT CRISIS

- **Leading Cause of Death:** Cardiovascular disease remains the world's leading cause of death, claiming over 20.5 million lives every year.
- **Subtle Beginnings:** Yet many serious cardiac conditions do NOT begin with dramatic symptoms.
- **The ASD Parallel:** As seen in our patient, a life-threatening defect can remain completely concealed until an incidental examination.

THE MOMENTS THAT MATTER

- A silent risk factor.
- A subtle symptom.
- A missed warning.
- A delayed consultation.
- Sometimes, these are the moments that decide a life.

The Cardiologist's Message: Do Not Wait for Heart Failure

Core clinical pillars for early cardiovascular protection and disease prevention

"As cardiologists, our message is simple: do not wait for the heart to fail before you start protecting it."

1. KNOW YOUR BIOMARKER NUMBERS

- **Vascular Screen:** Routinely monitor Blood Pressure to prevent silent hypertensive target-organ damage.
- **Metabolic Screen:** Check fasting Blood Glucose and complete Lipid Profile (LDL-C, HDL, Triglycerides).

2. RECOGNIZE SUBTLE RED FLAG SYMPTOMS

- **Never Ignore:** Do not dismiss unexplained chest discomfort, disproportionate breathlessness, or syncope.
- **Atypical Signs:** Investigate sudden palpitations, dizziness, or unusual chronic fatigue promptly.

3. LIFESTYLE FOUNDATIONS

- **Stop Tobacco:** Complete cessation of all tobacco and nicotine products.
- **Physical Activity:** Stay physically active with at least 150 minutes of moderate aerobic exercise weekly; maintain a healthy weight.

4. ADHERENCE & TIMELY CONSULTATION

- **Regular Compliance:** Take prescribed cardiovascular medications regularly and without self-discontinuation.
- **Early Action:** Seek immediate medical attention when warning signs appear—time is myocardium.

**“Because prevention is not merely about adding years to life —
it is about protecting the heart that makes life possible.”**

On this World Heart Day, let us listen more carefully —
to every symptom, every risk factor, and every heartbeat.

DON'T MISS A BEAT.

By The Numbers: Fascinating Realities of the Heart

Nine remarkable metrics capturing the astonishing physiology and history of cardiology

115,000 — Heartbeats Daily

The number of times the human heart beats in a single day.

2,000 Gallons — Blood Pumped Daily

Volume of blood circulated by the heart through the body each day.

1893 — First Heart Surgery

The year in which Dr. Daniel Hale Williams performed the first successful open-heart surgery.

3,500 Years — Earliest Recorded CVD

Age of the Egyptian mummy in which the earliest known case of heart disease was identified.

1,200 BPM — Fastest Heartbeat

The fastest mammalian heartbeat per minute — recorded in the tiny pygmy shrew.

1 Pound — Human Heart Weight

The average weight of the human heart, powering an entire lifetime of circulation.

60,000 Miles — Vascular Network

The total combined distance our blood vessel system can extend to if laid end-to-end.

1,500 Pounds — Blue Whale Heart

The weight of a blue whale's colossal heart, the largest organ of any living creature.

1.5 Gallons — Pumped Per Minute

The extraordinary volume of blood pumped by the human heart during each resting minute.



Thank You