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Severe Aortic Stenosis Concealed by a Negative History in Pregnancy: A Case Report Emphasizing the Diagnostic Cornerstones of History and Physical Examination

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ABSTRACT

Pregnancy concurrent with severe underlying cardiac disease presents a substantial risk to maternal and fetal well-being. Despite routine antenatal screening, patients may occasionally deny or be unaware of critical cardiac histories, complicating perioperative management and leading to catastrophic decompensation. We report the case of a 22-year-old primigravida admitted for labor with intrauterine fetal demise (IUFD). The initial history was non-contributory, and the physical examination was unremarkable. Following an uncomplicated delivery and regional analgesia, the patient experienced sudden hemodynamic collapse, seizure, and pulmonary edema, necessitating emergent intubation and intensive care. Subsequent echocardiography revealed previously undiagnosed severe aortic stenosis (AS) with a mean pressure gradient of approximately 100 mmHg. This case highlights how severe, compensated pathology can be masked by patient denial, underscoring the indispensable role of a meticulous history and physical examination as the primary diagnostic tool in obstetric anesthesia, regardless of self-reported symptoms.

Introduction

Pregnancy with underlying severe cardiac disease poses a significant risk to maternal and fetal outcomes [1,2]. The hemodynamic changes of pregnancy, including a 30–50% increase in plasma volume and cardiac output, significantly challenge the reserve of a diseased heart [3]. Although routine antenatal screening aims to identify such conditions, patients may occasionally conceal, minimize, or be unaware of critical medical histories, dramatically complicating perioperative management [4]. Given the time constraints and unique physiological stresses of the peripartum period, reliance on a comprehensive and meticulous history and physical examination is paramount [5]. We present a case emphasizing the importance of detailed history-taking

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and a high-suspicion physical examination in obstetric anesthesia, wherein a patient with concealed severe aortic stenosis experienced sudden, life-threatening decompensation post-delivery.

Case Presentation

A 22-year-old woman, primigravida, at 34 weeks and 1 day of gestation (based on last menstrual period and early ultrasound), was admitted for induction of labor following a diagnosis of intrauterine fetal demise (IUFD). No primary cause of IUFD, such as severe hypertension or infection, was found. The initial obstetric assessment was complicated by a history that was entirely non-contributory; the patient specifically denied any history of chronic illnesses, surgeries, familial diseases, or cardiac symptoms. Crucially, the physical examination was deceptively unremarkable, showing a regular rate and rhythm without murmurs or gallops, and the patient reported the ability to perform moderate physical activity without symptoms (NYHA Class I).

Anesthetic consultation was requested for labor analgesia. The patient was experiencing severe labor pain (Visual Analog Scale [VAS] 9). An epidural was performed at the L1–L2 interspace via a median approach using a loss-of-resistance technique. Following negative aspiration and a standard test dose, a mixture of 0.125% hyperbaric bupivacaine (10 mL) and fentanyl (25 mcg) was administered incrementally over ten minutes.

Continuous monitoring of vital signs showed stable parameters throughout the procedure, and the patient's pain score decreased to VAS 3 within 20 minutes. She delivered vaginally without complications at 13:30. Intravenous ondansetron (4 mg) and 1 L of Ringer's lactate were administered.

Approximately 2 hours and 35 minutes post-delivery, the patient experienced a sudden, catastrophic deterioration. The anesthesiology team was urgently called back as the patient exhibited a generalized seizure, upward gaze deviation, and respiratory distress with decreased consciousness, accompanied by tachycardia (initial HR 190 bpm) and hypoxia (see Figures 1–3). Given the sudden post-delivery symptoms, the first differential diagnosis from the obstetric perspective was eclampsia. The patient was given magnesium sulphate based on the clinical evaluation of the gynecologist and obstetrician at the onset of symptoms. Intravenous diazepam (10 mg) was administered immediately. The patient was promptly intubated for airway protection and respiratory failure using sodium thiopental and succinylcholine.

Table 1 shows the vital signs of the patient, and Table 2 illustrates the pharmacological decisions and their rationale. Initial laboratory results revealed severe mixed metabolic and respiratory acidosis (pH 6.94; PCO_2 79.9), elevated liver enzymes (ALT 6 U/L to 60 U/L), thrombocytopenia (platelets 127,000/µL to 90,000/µL), and a markedly elevated troponin (40,000 ng/L) (Table 3).

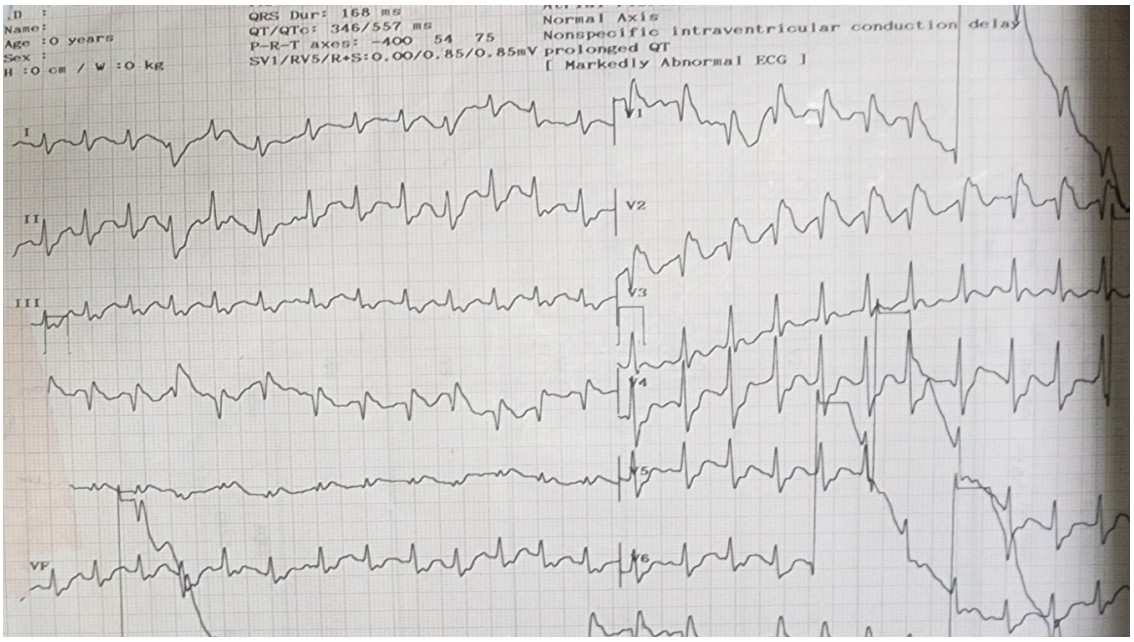

Fig. 1. EKG showing sinus tachycardia with a heart rate of 190 bpm, indicative of significant hemodynamic distress.

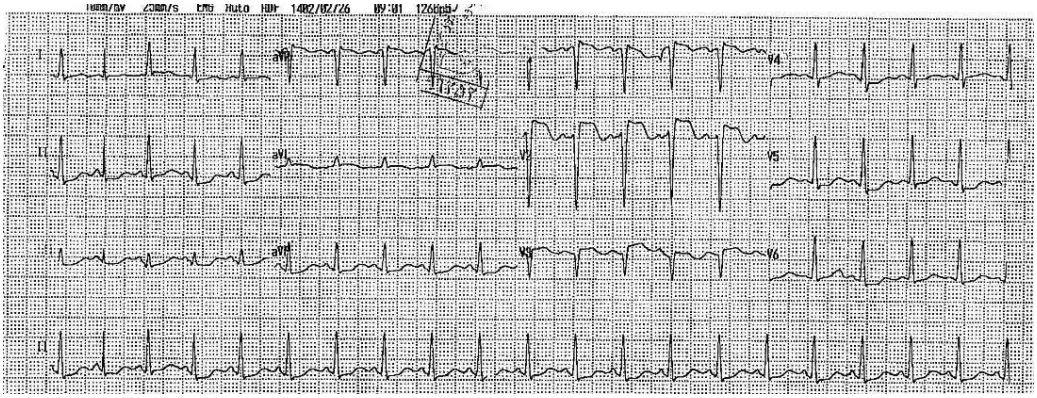

Fig. 2. EKG 3rd day, showing sinus tachycardia

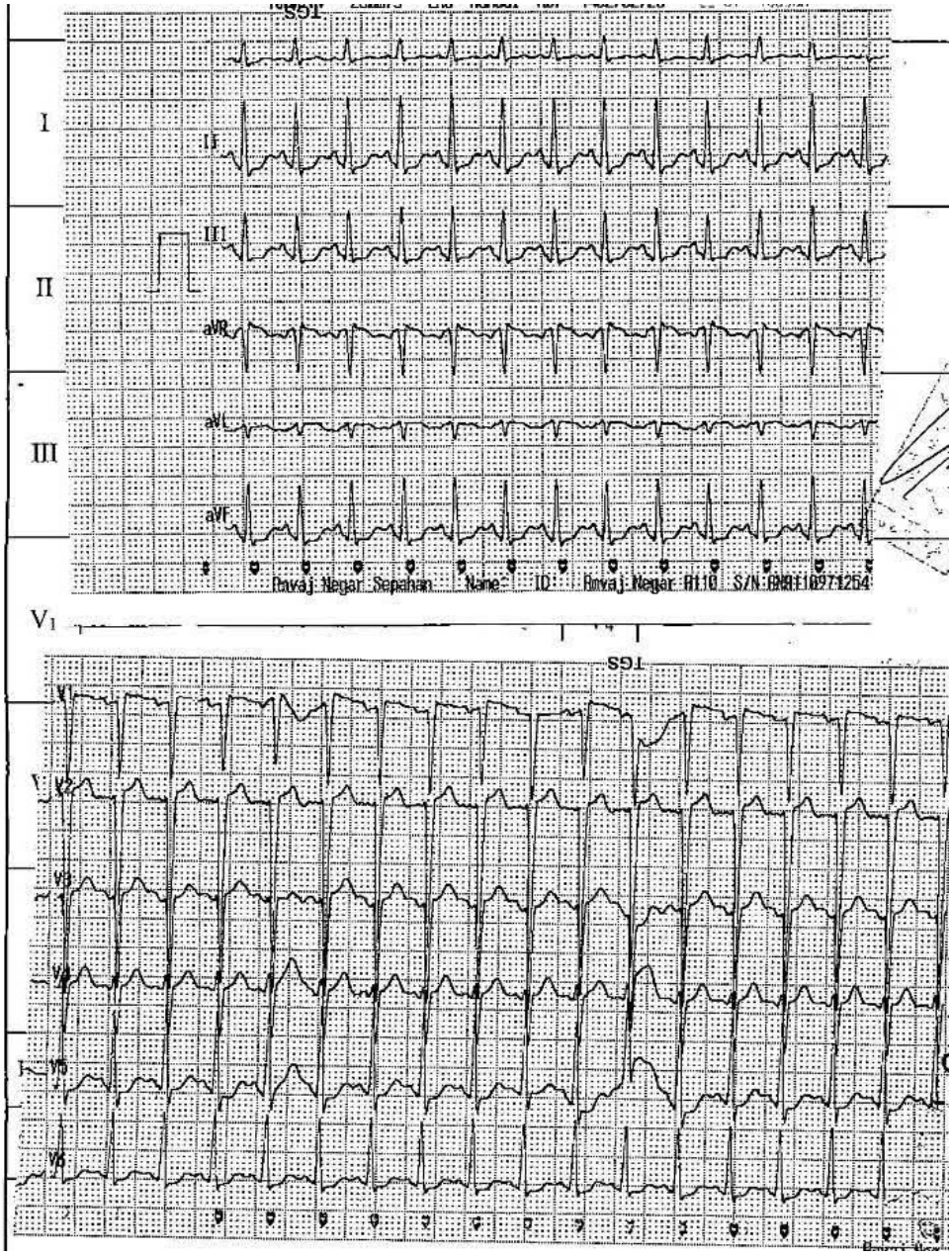

Fig. 3. EKG 5th day, showing sinus tachycardia

Table 1. The vital sign of the patient

VS	BP	T	HR	RR (spontaneous)	SPO_2	IVC DIAMETER	UO (cc)	GCS M V E	CVP (cmh20)
5 min after intubation	73/48	38	135	30	76	-	50	1 T 1	9
30 min after intubation	82/51	38	130	25	82	18mm	-	1 T 1	11
60 min after intubation	91/50	38	100	10	84	-	100	3 T 3	12
Next day	121/79(without inotropic drugs) *	37.2		8	98	-	100-130 cc/hour	6 T 4**	12
5 days later	122/83 without inotropic drugs) *	37		12-14	98	-	70cc/hour	6 5 4	12

Laboratory results revealed metabolic acidosis, elevated liver enzymes, thrombocytopenia, and markedly elevated troponin (40,000 ng/L). Ventilator settings were adjusted to SIMV volume mode with increasing FiO2 to maintain saturation.

Table 2. The pharmacological decisions and the reasons

administration	dose	Reason of starting
Dobutamine	1mic[1]/kg[2]/min[3]	HOTN[4]/doubt of heart failure
hydrocortisone	200mg[5]	Septic shock
frusemide	5mg/hour	Pulmonary edema/administrated if SBP[6] more than 90mmhg
amiodarone	150mg/in 20 min	VT[7] after intubation
norepinephrine	5-10 mic/min	If SBP less than 80 Despite the injection of Dobutamine
fentanyl	50 mic/q1 hour	If SBP more than 90 mmhg[8].
bicarbonate	200 meq (start after seeing second VBG)	100 meq[9] in 1 and 2nd hour.

1: microgram,2: kilogram.3: minute. 4: hypotension. 5: milligram. 6: systolic blood pressure. 7: ventricular tachycardia.8: millimeter of mercury.9: milli equivalent.

Pink frothy secretions and diffuse rales were noted bilaterally, consistent with acute pulmonary edema. Due to profound hypotension (BP 73/48 mmHg, 5 minutes post-intubation), a norepinephrine infusion was initiated, and a right internal jugular central venous line was placed under ultrasound guidance.

The differential diagnosis at this stage included catastrophic events common in the peripartum period: pulmonary embolism, acute heart failure, amniotic fluid embolism, intracranial mass, cerebrovascular events, drug poisoning, eclampsia, uterine rupture, uterine atony, and ARDS (adult respiratory distress syndrome).

She was transferred to the operating room adjacent to the delivery ward for intensive management. Due to hypotension, a norepinephrine infusion was initiated, and a right internal jugular central line was placed under ultrasound guidance.

On the following day, a cardiology consultation and transthoracic echocardiography identified previously undiagnosed severe aortic stenosis (AS) with a valve area of <1 cm^2, a peak velocity of 4.6 m/s, and a mean pressure gradient of approximately 100 mmHg. This severe fixed outflow obstruction explained the patient's critical deterioration under the hemodynamic stress of labor and the post-delivery fluid shift (Table 4).

The patient was transferred to a specialized cardiac center for multidisciplinary management. Following stabilization, she was extubated on day three and subsequently listed for heart transplantation. Unfortunately, post-discharge follow-up was lost, as the patient reportedly left the country.

Discussion

This case dramatically underscores the vital role of

Table 3. Initial laboratory results of patient in different times

Laboratory result	17pm	18pm	20 pm	Next day	Second day	Third day	Fifth day
Hb[1](g/dl)	12.9	11.4	12.3	10.1	-		11.9
Plt[2](ul)	127000	135000	121000	184000	-	158000	90000
PH	6.94	6.69	7.20	7.43	-	-	7.34
PCO2[4]	79.9	70	50.7	44			35
PO2[5]	61	56	78	88			37
HCO3[6]	16.3	10	22.6	24.7			24.9
BS[7]	213	-	-	179	-	-	113
WBC[8]	12800	22400	31500	18800	-	-	8800
BUN[9]	78	-	-	-	39	54	42
Cr[10]	0.8	1	-		0.8	0.8	1.2
ALT[11]	6	21	-	60	340	142	77
AST[12]	19	164	-	450	310	337	596
ALKP[13]	403	325	-	359	311	323	
LDH[3]	590	-	-	-	-		60
Na[14]	142	-	-	-	-	141	139
Troponin	40000	-	-	-	-		
potassium	4.3	-	-	2.7	3.9		
Ca[15]	-	9.9	-	8.6			8.3
Albumin	-	-	-	2.7	-	3.4	2.9
P[16]	-	5.2	-	2.7	2.4	-	
Bilirubin direct	-	0.9	-	0.5	0.6	0.6	
Bilirubin total		5.5	-	0.8	1.2	0.7	
PTT[17]	31	26	-	-	54	-	54
Fibrinogen	-	-	-			346	
Mg[18]			1.5				1.9

1: hemoglobin. 2: platelet. 3: lactate dehydrogenase. 4: pressure of carbonic dioxide. 5: pressure of oxygen. 6: bicarbonate. 7: blood sugar. 8: white blood cells. 9: blood urea nitrogen 10: creatinine.

11: alanine transaminase. 12: aspartate aminotransferase. 13: alkaline phosphate. 14: sodium. 15: calcium. 16: phosphorus. 17: partial thrombin time.18: magnesium.

Table 4. The patient's echocardiographic history during hospitalization

	1st day	2rd day	3rd day	20th day
Ejection fraction	50-55%	25-30%	10-15%	10%
AV peak velocity	4.6	4.6	4.6	4.6
AV mean PG	97	100	98	95
AVA (cm^2)	Less than 1	Less than1	Less than 1	Less than 1

comprehensive history-taking and a high-suspicion physical examination in obstetric anesthesia, particularly when patients may conceal critical medical information. Severe aortic stenosis (AS) is the most common hemodynamically significant valvular lesion in pregnancy, carrying a high risk of maternal mortality and morbidity [6].

Severe aortic stenosis is a rare but life-threatening condition during pregnancy [7]. While the patient reported no history and exhibited a seemingly normal physical examination (NYHA Class I), the underlying fixed cardiac obstruction was critically susceptible to two key hemodynamic changes:

· **Volume Shifts:** The immediate post-delivery state involves a rapid auto-transfusion of blood from the contracting uterus, suddenly increasing venous return. In AS, the left ventricle is hypertrophied and cannot handle acute volume changes, leading to elevated left ventricular end-diastolic pressure, rapid development of pulmonary edema, and subsequent hypoxia and seizures (secondary to cerebral hypoperfusion) [8]. The increased preload can precipitate heart failure in the setting of fixed cardiac output [7].

· **Tachycardia:** The patient's initial tachycardia (HR:190 bpm) is disastrous in AS. The hypertrophied ventricle relies heavily on a long diastolic filling time; tachycardia drastically shortens this period, leading to a catastrophic drop in cardiac output and myocardial ischemia (evidenced by the massive troponin elevation) [9].

The finding of severe AS after the event highlights a critical diagnostic gap. While the patient denied symptoms, a meticulous physical examination may have detected a subtle, though often absent, systolic murmur or other peripheral signs of heart failure [10]. The rapid evaluation conducted under time pressure,

while understandable, likely contributed to accepting the patient's self-reported non-contributory history without an extensive, focused cardiac examination (i.e., auscultation in different positions, checking for *pulsus parvus et tardus*). It is well established that even asymptomatic patients with severe AS may decompensate dramatically during the physiological stress of labor and delivery [8,9].

We acknowledge the inherent difficulty in auscultating for aortic stenosis during pregnancy, as physiological changes (e.g., hyperdynamic circulation, positional changes) can mask or diminish murmurs. However, this case reinforces the need for high clinical suspicion, as subtle findings—such as a faint murmur, a slightly delayed carotid upstroke, or unexplained mild dyspnea—can serve as critical hints for underlying disease, even when the patient is seemingly asymptomatic [10]. The American Heart Association guidelines emphasize the necessity of a thorough cardiac evaluation in any pregnant patient with unexplained symptoms or abnormal physical findings [7].

This case serves as a powerful reminder: the anesthetic evaluation cannot simply accept a patient's denial of symptoms. Clinicians must maintain a high index of suspicion, especially in the setting of unexplained hemodynamic instability, and conduct a thorough, focused physical examination that is repeated if there is any change in status [4,5].

A limitation of this case report is the reliance on patient-reported history, which was ultimately found to be misleading. Furthermore, the loss of follow-up prevents a complete assessment of the patient's long-term outcome.

Conclusion

In obstetric anesthesia, a thorough history and physical examination remain the cornerstone of safe care and the most powerful non-invasive diagnostic tools. Concealed or undiagnosed severe cardiac diseases, such as aortic stenosis, can be masked by patient denial and compensation, only to present suddenly with life-threatening complications following the hemodynamic shifts of delivery. This case emphasizes the non-negotiable need for clinical vigilance and a high index of suspicion; the initial physical examination must be meticulous, and subtlety should never be equated with normality when faced with life-or-death decision-making. Multidisciplinary approaches and timely, goal-directed interventions are critical to optimize maternal outcomes once decompensation occurs. This case serves as a crucial reminder to evaluate maternal heart status, even in seemingly healthy obstetric patients, especially those undergoing delivery, given the potential for rapid cardiovascular decompensation.

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Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors have no conflict of interest to declare.

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