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| RESEARCH ARTICLE

Back Pain Concealing a Life-Threatening Normotensive Aortic Dissection in a Pregnant Lady

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| ABSTRACT

The absence of hypertension should not be considered sufficient to rule out aortic dissection. In fact, clinical characteristics such as the type, intensity, and sudden onset of symptoms often yield greater diagnostic insight than isolated blood pressure values. During pregnancy, normal physiological adaptations can mask or mimic critical conditions like aortic dissection, further complicating timely diagnosis. This case report illustrates that principle through the presentation of a 32-year-old woman, gravida 2 para 1, at 28 weeks of gestation, who arrived at the emergency department with acute interscapular back pain of three hours' duration. She subsequently developed intermittent hypotension, prompting advanced imaging with chest radiography, transesophageal echocardiography, and magnetic resonance aortography, which collectively confirmed the diagnosis of a Stanford type A aortic dissection associated with pregnancy. The patient underwent emergent surgical repair with favorable maternal and fetal outcomes, and a long-term surveillance plan was established. The case underscores ongoing uncertainties in the literature regarding optimal clinical strategies during the "gray zone" of gestational age—specifically between 28 and 32 weeks—where standardized guidance is lacking.

| KEYWORDS

Aortic Dissection, Pregnancy, Back pain, Hypertension, Maternal Mortality.

| ARTICLE INFORMATION

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1. Introduction

Acute aortic dissection (AD) is a rare but highly lethal condition. Autopsy studies suggest a prevalence of 1–3% for undiagnosed dissections, and registry data estimate an annual incidence on the order of 5–30 per million persons (roughly 1–6 per 100,000) [1]. In the general population, untreated AD is catastrophic – mortality rises ~1% per hour after onset, reaching ~50% by 48–72 hours [1]. Globally, contemporary reports quote in-hospital or 30-day mortality of 20–30% for Stanford type A dissections involving the ascending aorta, and ~10% for type B (descending only) when managed appropriately [2]. Acute AD is more common in older men, but when it occurs in young women or during pregnancy, the stakes are especially high. In pregnancy,

the incidence is exceedingly low – on the order of 4–5 cases per million pregnancies [3] – but pregnancy-related dissections account for a disproportionate share of aortic catastrophes in women. For example, AD complicates only about 0.01%–0.04% of pregnancies [3] [4], yet it is one of the top cardiovascular causes of maternal mortality. Historical series report maternal death rates up to 30%, with fetal loss in 20–50%. Modern series show improved outcomes (~6–10% maternal mortality, ~10–20% fetal loss) due to earlier recognition and management [3]. Reliable data specific to the Middle East are lacking, but available global estimates suggest regional incidence and risk factor patterns broadly mirror those in Western cohorts. Several risk factors underlie most AD events. Systemic hypertension is by far the most common predisposing condition, noted in roughly 70–75% of patients overall [5]. Other contributors include atherosclerotic aortic aneurysms, prior cardiac surgery, and inflammatory or traumatic aortic injury. For younger patients, hereditary aortopathies also feature prominently — e.g., congenital anomalies such as bicuspid aortic valve, aortic coarctation, and Turner syndrome, as well as connective-tissue disorders like Marfan syndrome, Loeys–Dietz, and vascular Ehlers–Danlos — all of which create weakened aortic walls prone to dissection under stress [3]. In women of childbearing age, pregnancy itself is a potent precipitant. Physiological changes in pregnancy — expanding blood volume, increased cardiac output and heart rate, reduced systemic vascular resistance, and high levels of estrogen and progesterone — markedly increase aortic wall stress [3]. These alterations peak in the late third trimester and early postpartum, coinciding with the majority of peripartum dissections. In fact, pregnancy raises the risk of AD by roughly 4- to 25-fold compared to non-pregnant women [3]. Histologic studies of pregnant aortas document media degeneration — loss of elastic fibers, mucopolysaccharides, and other changes — consistent with hormone-mediated weakening [3]. Chronic hypertension or hypertensive crises, including preeclampsia, further amplify this risk. A family history of aortic events or a known aortic aneurysm are also strong red flags. Notably, a modest minority of AD patients have no history of hypertension; in large series, about 25–30% of dissections occur in people without previously diagnosed high blood pressure [5]. Clinically, acute AD usually presents sudden, severe pain described as tearing or ripping in the chest, back, or abdomen. Chest pain is by far the most common symptom: in broad registry data, roughly 60–70% of dissections report abrupt chest pain [2]. Back pain also occurs but less frequently — perhaps 15–30% of cases overall, more often in type B dissections where it can be the predominant symptom [2]. Pure abdominal pain or other atypical symptoms — e.g., neurologic deficits, syncope, or visceral ischemia — are less common. However, practitioners must remain alert because AD can masquerade. For example, one series reported that 77.5% of dissection patients had some back pain, and 45% had isolated back pain without chest discomfort [6]. Such presentations are easily misinterpreted as musculoskeletal or obstetric backache. In pregnancy, normal changes — such as back strain, reflux, and musculoskeletal pains — and a cautious attitude toward invasive testing often delay consideration of AD. Classical chest pain may be absent, and weaker or atypical symptoms — e.g., back or abdominal pain, dyspnea, or syncope — are reported in some cases [4] [6]. Because rapid diagnosis is critical, imaging strategy in pregnancy must balance speed and safety. Transthoracic or transesophageal echocardiography is generally the initial modality of choice in a hemodynamically unstable patient. It is widely available at the bedside and contains no radiation. Echocardiography can detect proximal dissections, aortic regurgitation, tamponade, or pericardial effusion. MRI (without gadolinium) is considered safe for the fetus and provides excellent aortic views, but its use is often limited by availability and the time required. Computed tomographic angiography (CTA) is the reference standard in non-pregnant patients for delineating the intimal flap, true/false lumen, and branch involvement. In pregnancy, fear of fetal radiation frequently gives pause, but studies note that modern CT doses — especially in the third trimester — are below teratogenic thresholds [4]. Nonetheless, clinicians typically reserve CTA for cases of high suspicion when other modalities are inconclusive [4]. These imaging concerns can lead to diagnostic delays. Surveys of pregnancy-related AD emphasize that investigators often defer CT or await delivery out of caution, paradoxically worsening outcomes. Delayed recognition of AD is dangerous. Every hour of untreated proximal (type A) dissection increases mortality by roughly 1% [1]. Even with optimal management, outcomes depend heavily on promptness — late presenters have far higher complication and death rates. In the general population, reported 30-day mortality for treated type A AD ranges ~20–30% [2], and any misdiagnosis can lead to catastrophic extension or rupture before therapy. In pregnancy, delays compound risks to both mother and fetus. Studies have long shown that women with AD diagnosed only at rupture or at surgery have dramatically worse survival. In contrast, early detection and surgical or endovascular repair can reduce maternal death toward single digits [3]. Taken together, these features underscore why the present case is noteworthy. The patient had only mild gestational hypertension — well below levels usually seen in AD — and no prior aortic disease. Her only symptom was acute back pain, without the classic tearing chest pain that typically triggers alarm. In isolation, such back pain in pregnancy more often suggests benign causes like muscle strain or disc herniation, so a high index of suspicion was required to pursue advanced imaging. In summary, this case differs from typical AD in pregnancy in having only trivial hypertension and an atypical presentation of isolated back pain — a combination that concealed the life-threatening diagnosis.

2. Case Presentation

2.1 Patient's history and Physical Examination

We report the case of a 32-year-old gravida 2, para 1 woman at 28 weeks' gestation who presented to the emergency department with a primary complaint of acute, severe back pain persisting for the preceding three hours. The pain had an abrupt onset, was localized to the interscapular region with radiation to the lumbar spine and was characterized by the patient as a

tearing sensation—qualitatively distinct from her prior musculoskeletal discomfort. She denied any history of trauma. There was no alleviation of symptoms with changes in position, and she reported no associated vaginal bleeding, uterine contractions, fever, or rupture of membranes. Additionally, she denied chest pain, syncope, seizures, or any focal neurological deficits, although she did endorse mild nausea and non-exertional dyspnea. Her past medical and surgical history were non-contributory, as was her obstetric history, with a previous pregnancy course described as uncomplicated. She had no history of hypertension, diabetes mellitus, or connective tissue disorders. A family history was notable for sudden cardiac death in her father at the age of 52. The patient reported taking only prenatal vitamins and oral iron supplementation. Social history was negative for tobacco use, alcohol consumption, or recreational drug use. On physical examination, the patient appeared visibly anxious and in acute pain. Vital signs on presentation revealed tachycardia (heart rate: 108 bpm), tachypnea (respiratory rate: 24 breaths per minute), normothermia (temperature: 36.8°C), and normoxia with oxygen saturation of 96% on ambient air. She was normotensive, with a right arm blood pressure measurement of 118/72 mmHg. Cardiopulmonary and abdominal examinations were unremarkable. Peripheral pulses, including bilateral radial and lower extremity pulses, were symmetrically palpable. The gravid uterus was consistent with a gestational age of 28 weeks, non-tender to palpation, without evidence of uterine contractions. Fetal heart tones were auscultated at 150 bpm with a regular rhythm.

2.2 Investigations

Electrocardiography demonstrated sinus tachycardia without evidence of ischemic changes. Relevant laboratory investigations are summarized in Table 1. As the patient developed hemodynamic instability and hypotension, accompanied by intermittent episodes of chest discomfort, a chest radiograph with appropriate fetal shielding was obtained following informed consent. The imaging revealed a widened mediastinum and a small left-sided pleural effusion, findings that raised concern for aortic dissection. A transesophageal echocardiogram (TEE) was subsequently performed, which demonstrated an intimal flap within the ascending aorta extending into the aortic arch, along with moderate aortic regurgitation—findings consistent with an acute Stanford type A aortic dissection. Further evaluation with magnetic resonance imaging (MRI) aortography confirmed a type A dissection extending from the ascending aorta through the proximal descending thoracic aorta, without involvement of the abdominal aorta.

Test	Result	Normal Range
Hemoglobin	11.4	12-16 g\dL
WBC	$8.2x10^9$	$4.0\text{-}11x10^9$\L
Platelets	$230x10^9$	$150\text{-}450x10^9$\L
Sodium	136	135-145 mmol\L
Potassium	4.2	3.5-5.0 mmol\L
Creatinine	0.7	0.7-1.2 mg\dL
ALT	WNL	<40 U\L
AST	WNL	<40 U\L
Troponins	<0.1	<0.1 ng\mL

Table 1: results of relevant laboratory investigations.

3. Management course

Due to subsequent recurrent episodes of hypotension, resuscitative measures were promptly initiated. Supplemental oxygen was administered via face mask, targeting an SpO_2 >95%. A left lateral tilt was applied to mitigate aortocaval compression, with continuous maternal cardiac and fetal monitoring maintained throughout. Antihypertensive therapy was avoided, as the patient remained normotensive to mildly hypotensive; instead, judicious fluid resuscitation was initiated with a 250 mL crystalloid bolus. Intravenous morphine was carefully titrated for analgesia. Following intervention, the heart rate stabilized within the range of 70–75 beats per minute, and systolic blood pressure was maintained around 110 mmHg. Upon radiological confirmation of the diagnosis, a multidisciplinary team comprising obstetricians and cardiothoracic surgeons was promptly activated. Emergent surgical intervention was undertaken, consisting of ascending aortic graft replacement via median sternotomy under cardiopulmonary bypass. Intraoperative monitoring included continuous fetal heart rate surveillance, with the fetus remaining stable throughout the procedure. The moderate aortic regurgitation was corrected intraoperatively. Total cardiopulmonary bypass time was 120 minutes. Postoperatively, the patient required initial mechanical ventilation and sedation in the intensive care unit, followed by successful extubation on postoperative day one. Hemodynamic stability was maintained with intravenous fluids alone, without the need for vasopressors or antihypertensive agents. Oral labetalol 100 mg twice daily was initiated once the patient achieved clinical stability and normotensive readings, in order to prevent any stress on the wall of the aorta. Serial transthoracic echocardiograms demonstrated a stable graft with preserved left ventricular ejection fraction. The patient was discharged on postoperative day seven on oral beta-blocker therapy and was classified as high-risk for future pregnancies,

warranting close obstetric and lifelong cardiovascular follow-up. Both the mother and fetus were clinically stable at the time of discharge.

4. Discussion

In pregnancy, symptoms such as back pain, chest discomfort, and dyspnea are frequently misattributed to musculoskeletal or obstetric etiologies, leading to delays in the recognition of life-threatening conditions such as aortic dissection [1]. This underscores the critical importance of physician awareness regarding red flag features associated with back pain—particularly when characterized by sudden onset, severe tearing quality, associated diaphoresis, dyspnea, or the presence of a new cardiac murmur [2]. Notably, 12–15% of aortic dissections may present in the absence of hypertension, further obscuring timely diagnosis [2]. Normotension in the context of aortic dissection can occur when perfusion through the true lumen is compromised, resulting in reduced effective stroke volume and, consequently, lower systemic blood pressure [2]. Pregnancy itself is a recognized risk factor for aortic dissection, primarily due to hormonally mediated hemodynamic alterations, including a 40–50% increase in circulating blood volume, a 30–50% rise in cardiac output, and a physiological elevation in heart rate by 10–20 beats per minute [4]. These changes contribute significantly to increased wall stress within the aorta, with the majority of dissections occurring during the third trimester or early postpartum period [2]. While chest radiography offers approximately 60% sensitivity for identifying potential aortic dissection and serves as a rapid, albeit non-definitive, initial screening modality, its use during pregnancy is limited due to concerns regarding fetal radiation exposure [7]. Similarly, computed tomography angiography (CTA), though the diagnostic gold standard in non-pregnant patients, is associated with ionizing radiation and thus carries fetal risk [7]. Transesophageal echocardiography (TEE), with a reported sensitivity and specificity of 95% for proximal dissections, represents a valuable and rapid diagnostic tool in hemodynamically unstable patients [7]. However, complete visualization of the thoracic and abdominal aorta in pregnant patients often necessitates magnetic resonance aortography, which avoids ionizing radiation and remains the preferred modality for comprehensive assessment in this population [7]. Pulmonary embolism (PE) should remain a prominent differential diagnosis in such clinical scenarios, despite the nature of the patient's symptoms—namely, abrupt, tearing pain rather than sudden pleuritic chest pain—being more suggestive of dissection. Nonetheless, PE cannot be definitively excluded without appropriate diagnostic evaluation, as overlapping features may lead to diagnostic ambiguity. Strong clinical indicators such as pulse deficits or inter-arm blood pressure discrepancies may help distinguish aortic dissection from PE but are not always present [7]. In complex decision-making scenarios such as this, maternal stabilization must be prioritized, as maternal mortality almost invariably results in fetal demise [2]. Maternal mortality rates for pregnancy-associated Stanford type A aortic dissection are reported at 20–30%, rising to nearly 50% in the absence of surgical intervention [1]. Fetal mortality ranges from 20–25%, increasing significantly in the presence of maternal shock [1]. According to the 2022 International Registry of Acute Aortic Dissection (IRAD), mortality in untreated type A dissection increases by 1–2% with each passing hour. Delivery should be considered when gestational age is viable and maternal hemodynamic instability is profound, further complicating management in cases such as this, given the fetal maturity at 28 weeks [3]. In pregnancies beyond 32–34 weeks' gestation with maternal stability, emergent cesarean delivery prior to surgical repair of the dissection may improve neonatal outcomes [3]. However, in the gray zone between 28 and 32 weeks, clinical decisions must be individualized, balancing maternal and fetal risks, and highlighting existing gaps in the literature regarding optimal timing and sequencing of interventions. In normotensive patients with confirmed aortic dissection, intravenous beta-blockade is contraindicated; however, low-dose oral beta blockers are still recommended in the postoperative setting for aortic wall protection and improved long-term survival, irrespective of baseline blood pressure, in accordance with the 2022 European Society of Cardiology (ESC) guidelines. Following surgical repair, lifelong beta-blocker therapy and surveillance with transthoracic echocardiography every 6–12 months are indicated [7]. Genetic evaluation for connective tissue disorders, including Marfan syndrome and Loeys-Dietz syndrome, should also be undertaken [7]. Given the significant clinical overlap between benign pregnancy-related symptoms and red flags for catastrophic pathologies such as aortic dissection, compounded by diagnostic limitations imposed by fetal safety considerations, clinicians must maintain a high index of suspicion. A proactive, action-oriented diagnostic approach should be favored over one of omissions, as early recognition and timely intervention are essential to optimizing maternal and fetal outcomes.

5. Conclusion

Normotensive blood pressure readings should never exclude the diagnosis of aortic dissection. The nature, quality, and abruptness of symptom onset often provide more diagnostic value than numerical blood pressure measurements alone. Furthermore, the physiological changes of pregnancy can obscure or mimic the presentation of life-threatening conditions such as aortic dissection. Therefore, symptoms like chest pain, dyspnea, or back pain should not be reflexively attributed to musculoskeletal or obstetric causes, particularly when red flag features—such as a new murmur, tearing pain quality, or sudden onset—are present. While chest radiography remains a common initial screening tool for suspected aortic dissection, its use during pregnancy is understandably limited due to fetal radiation concerns. However, these risks must be weighed against the potential diagnostic benefit in time-sensitive scenarios, and its use should not be categorically excluded when clinical suspicion is high. This case also highlights persistent gaps in the literature regarding clinical decision-making in "gray zones" of gestational

age—particularly between 28 and 32 weeks—where the optimal sequencing of interventions remains unclear. Specifically, it remains a clinical dilemma whether to prioritize maternal survival through immediate surgical repair or to perform cesarean delivery first to optimize fetal outcomes in relatively stable patients. In such contexts, individualized decision-making remains essential, guided by multidisciplinary input and patient-specific factors.

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