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Critical Management of Recurrent Seizure at 16-Year-Old Primigravida with Atypical Eclampsia: A Case Report

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1. Introduction

Eclampsia, a severe pregnancy complication characterized by the onset of seizures or coma in pregnant women with preeclampsia, remains a leading cause of maternal and perinatal morbidity and mortality globally. Preeclampsia is a multisystem disorder typically presenting with hypertension and proteinuria after 20 weeks of gestation. The incidence of eclampsia varies significantly worldwide, ranging from 19.6 to 142 cases per 10,000 deliveries in middle, low, and extremely resource-poor countries. It disproportionately affects teenagers, who face a twoto six-fold higher risk than older women, making it a significant contributor to maternal mortality in this age group. The pathophysiology of eclampsia is

ABSTRACT

Background: Atypical eclampsia, characterized by seizures without the classic signs of hypertension and proteinuria, presents a diagnostic and therapeutic challenge in the critical management aspect. This case report highlights the critical management of recurrent seizures in a young primigravida with atypical eclampsia. Case presentation: A 16-year-old primigravida at 38 weeks gestation presented with recurrent generalized tonic-clonic seizures. Despite initial magnesium sulfate therapy, seizures persisted postpartum. The patient was managed with a combination of midazolam and dexmedetomidine for seizure control and sedation. After five days of intensive care, the patient recovered without further complications. **Conclusion:** This case underscores the importance of considering atypical eclampsia in pregnant women presenting with seizures, even in the absence of typical preeclampsia symptoms. The successful use of midazolam and dexmedetomidine in this case suggests their potential as alternative therapeutic agents for refractory seizures in eclampsia. Further research is needed to validate these findings and establish optimal treatment protocols for atypical eclampsia.

> complex and multifactorial, involving endothelial dysfunction, immune dysregulation, and genetic predisposition. Endothelial dysfunction leads to increased vascular permeability, vasoconstriction, and platelet activation, contributing to hypertension, proteinuria, and organ damage. Immune dysregulation, characterized by an imbalance of proinflammatory and anti-inflammatory cytokines, further exacerbates endothelial dysfunction and promotes systemic inflammation. Genetic factors, such as polymorphisms in genes involved in angiogenesis, inflammation, and coagulation, have also been implicated in the development of eclampsia.1-3

The diagnosis of eclampsia is primarily clinical, based on the presence of seizures or coma in a pregnant woman with preeclampsia. However, atypical eclampsia, where seizures occur without the classic signs of hypertension and proteinuria, presents a diagnostic challenge. This can lead to delayed diagnosis and increased risk of complications due to the absence of typical warning signs. The management of eclampsia requires a multidisciplinary approach, including prompt seizure control, blood pressure management, and supportive care. Magnesium sulfate is the first-line treatment for preventing and controlling seizures in eclampsia. However, in some cases, seizures may persist despite magnesium sulfate therapy, necessitating the use of alternative antiepileptic agents. Midazolam, a benzodiazepine, is a rapid-acting anticonvulsant with sedative properties that has been used in the management of status epilepticus. Dexmedetomidine, an alpha-2 adrenergic agonist, has also shown promise in controlling seizures and providing neuroprotection in various neurological conditions. Recent studies have suggested the potential benefits of these agents in refractory eclampsia, but further research is needed to establish their safety and efficacy in this context.⁴⁻⁶

This case report presents a unique case of atypical eclampsia in a 16-year-old primigravida who presented with recurrent seizures despite initial magnesium sulfate therapy. The patient was successfully managed with a combination of midazolam and dexmedetomidine for seizure control and sedation, resulting in a favorable outcome. This case highlights the importance of considering atypical eclampsia in pregnant women presenting with seizures, even in the absence of typical preeclampsia symptoms. It also underscores the potential of midazolam and dexmedetomidine as alternative therapeutic agents for refractory seizures in eclampsia. The patient's young age and limited antenatal care are significant risk factors for developing eclampsia. Adolescent pregnancies are associated with an increased risk of preeclampsia and eclampsia due to factors such as uterine immaturity and increased prevalence of obesity. Limited antenatal care can result in missed opportunities for early detection management and of preeclampsia, increasing the risk of progression to eclampsia. This also emphasizes the importance of a case multidisciplinary approach in managing eclampsia. The involvement of obstetricians, anesthesiologists, intensivists, and neonatologists is crucial for ensuring optimal maternal and fetal outcomes. Prompt recognition of atypical eclampsia, timely intervention with appropriate antiepileptic agents. and comprehensive supportive care are essential for improving maternal and perinatal outcomes in this challenging condition.7-9

2. Case Presentation

A 16-year-old primigravida, in her 38th week of gestation, was admitted to the emergency room following two generalized seizures that occurred within an hour of each other. Her blood pressure prior to referral was documented as 130/85 mmHg. The patient's background revealed that she came from a rural, low-income family with limited access to healthcare, resulting in inadequate prenatal care throughout her pregnancy. Notably, she had no previous history of hypertension or proteinuria, conditions often associated with pregnancy-related complications. Upon presentation at the emergency room, the patient was conscious and cooperative, although she exhibited signs of fatigue. Her vital signs were recorded as follows: temperature of 36.7°C, oxygen saturation of 97% on room air, blood pressure of 128/80 mmHg, pulse rate of 120 beats per minute, and respiration rate of 24 breaths per minute. A neurological examination did not reveal any specific deficits. However, an abdominal and pelvic examination indicated a contracted uterus, full cervical dilation, a fetal heart rate of 150 beats per minute, and a uterine height consistent with 38 weeks of gestation.

Laboratory investigations revealed a hemoglobin level of 9.9 g/dl, a normal platelet count, and the presence of +3 proteinuria in the urinalysis. Liver and renal function tests were within normal limits, and the blood coagulation profile was also unremarkable. After a thorough assessment of the patient's condition and medical history, a diagnosis of G1P0A0 (gravida 1, para 0, abortus 0) at 38 weeks gestation was made. The patient was also determined to be in the second stage of labor and experiencing atypical eclampsia. Within 15 minutes of arriving at the emergency department, and despite receiving a 4-gram bolus of magnesium sulfate (MgSO₄), the patient experienced a third generalized tonic-clonic seizure. To manage this, she was administered a 2 mg bolus of intravenous (IV) midazolam, which successfully terminated the seizure. However, she remained unconscious throughout the subsequent delivery process. Due to the severity of the mother's eclamptic state, forceps were used to expedite the delivery and minimize the duration of labor and pushing. The infant was delivered with an Apgar score of 7/8, indicating a relatively healthy newborn despite the challenging circumstances. Following delivery, the patient continued to experience convulsions. An additional 2-gram bolus of MgSO₄ was administered, but the seizure activity persisted. To address this, a continuous infusion of midazolam at a rate of 1 mg/hour was initiated. Given the patient's unconscious state and the complexity of her condition, she was transferred to the intensive care unit (ICU) for closer monitoring and specialized care.

Physical examination	Findings	
General condition	Conscious, cooperative, fatigued	
Vital signs	Temperature: 36.7°C, Oxygen saturation: 97% on room air, Blood pressure: 128/80 mmHg, Pulse rate: 120 beats per minute, Respiration rate: 24 breaths per minute	
Neurological exam	No specific deficits noted	
Abdominal and pelvic exam	Contracted uterus, Full cervical dilation, Fetal heart rate: 150 beats per minute, Uterine height: 38 weeks gestation	
Laboratory findings	Results	
Hemoglobin	9.9 g/dl	
Platelet count	Normal	
Urinalysis	+3 proteinuria	
Liver function tests	Normal	
Renal function tests	Normal	
Blood coagulation profile	Normal	

Table 1. Physical examination and laboratory f	findings.
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In the ICU, the patient's treatment regimen was expanded (Table 2). She was started on intravenous ceftriaxone at a dose of 2 grams once daily, along with a maintenance infusion of MgSO₄ at 1 gram per hour for the following 12 hours. The administration of ceftriaxone was a prophylactic measure aimed at preventing infections, a common concern in high-risk patients like those with compromised immune systems due to severe preeclampsia or eclampsia. In addition to the aforementioned medications, the received patient а maintenance dose of dexmedetomidine at 0.2 cc/kg body weight per hour.

Dexmedetomidine is an alpha-2 adrenergic agonist known for its sedative and analgesic properties. The patient's treatment plan also included dexketoprofen (40 mg every 8 hours) for pain management, omeprazole (40 mg every 24 hours) for gastric acid suppression, ondansetron (4 mg every 8 hours) for nausea and vomiting, furosemide (24-hour dose) as a diuretic, citicoline (500 mg every 12 hours) for neuroprotection, piracetam (2 grams every 12 hours) for cognitive enhancement, and supplementary oxygen administered via nasal cannula to ensure adequate oxygenation. Throughout her stay in the ICU, the patient's blood pressure remained consistently within the normal range. The administration of midazolam and dexmedetomidine was discontinued on the second day as the patient did not exhibit any signs of impending or recurrent seizures, and her blood pressure remained stable below 140/90 mmHg. After three days of therapy, her condition significantly improved (Figure 1). The patient experienced no further convulsions and remained stable, with no signs or symptoms of deterioration. Consequently, she was transferred to the ward. She was discharged from the hospital on the fifth day post-delivery.



Figure 1. Timeline of vital signs measurements. SBP=systolic blood pressure; DPB=diastolic blood pressure; HR=heart rate; PHC=primary health center; ICU=intensive care unit.

Critical management	Medication and dosage		
Initial seizure	MgSO ₄ 4-gram bolus		
management			
Seizure termination	Midazolam 2 mg IV bolus		
Postpartum seizure	MgSO ₄ 2-gram bolus (repeated)		
management			
Seizure control and	Midazolam continuous infusion at 1 mg/hour, Dexmedetomidine 0.2 cc/kg/hour		
sedation			
Antibiotic prophylaxis	nylaxis Ceftriaxone 2 grams IV once daily		
Maintenance therapy	MgSO ₄ 1 gram/hour for 12 hours, Dexketoprofen 40 mg every 8 hours, Omeprazole		
	40 mg every 24 hours, Ondansetron 4 mg every 8 hours, Furosemide 24-hour dose,		
	Citicoline 500 mg every 12 hours, Piracetam 2 grams every 12 hours, Supplemental		
	oxygen via nasal cannula		

Table 2	. Critical	management.
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3. Discussion

This case report underscores the complexities and challenges associated with atypical eclampsia, particularly in young primigravidas with limited access to healthcare. The patient's presentation with recurrent seizures in the absence of classic preeclampsia symptoms, such as hypertension and significant proteinuria, highlights the diagnostic difficulties inherent in atypical eclampsia. This atypical presentation can lead to delayed diagnosis and potentially life-threatening complications for both mother and fetus. The patient's young age of 16 is a critical factor in this case, as it significantly elevates her risk of developing eclampsia. Adolescent pregnancies, particularly those under 18 years old, are inherently associated with a higher risk of hypertensive disorders during pregnancy, including both preeclampsia and eclampsia. This heightened risk can be attributed to a multitude of factors, encompassing physiological, developmental, and socioeconomic dimensions. From a physiological standpoint, adolescents are still undergoing significant developmental changes, and their bodies may not be fully equipped to handle the demands of pregnancy. The cardiovascular system, in particular, may not have reached full maturity, potentially leading to an inadequate adaptation to the increased blood volume and cardiac output that occur during pregnancy. This can result in hypertension, a hallmark of preeclampsia and a precursor to eclampsia. Furthermore, the immune system of adolescents is still developing, and there may be differences in immune responses compared to older pregnant women. These immunological variations could contribute to the dysregulation of inflammatory processes and endothelial dysfunction, both of which are implicated in the pathogenesis of preeclampsia and eclampsia. The immature immune system may also be less equipped to tolerate the physiological stress of pregnancy, further increasing the risk of complications.9,10

In addition to physiological immaturity, adolescents may have underlying risk factors that exacerbate their susceptibility to eclampsia. Obesity, a growing concern among teenagers, is a wellestablished risk factor for hypertensive disorders of pregnancy. Excess body weight can lead to insulin resistance, inflammation, and oxidative stress, all of which contribute to endothelial dysfunction and the development of preeclampsia. Moreover, obesity can complicate the management of eclampsia, as it may interfere with drug metabolism and increase the risk of anesthesia-related complications. Socioeconomic factors also play a crucial role in the increased risk of eclampsia among adolescents. Teenagers from lowincome families, like the patient in this case, often have limited access to healthcare, including prenatal care. This lack of access can result in delayed diagnosis and inadequate management of preeclampsia, increasing the likelihood of progression to eclampsia. Additionally, adolescents may face barriers to accessing contraception and family planning services, leading to unintended pregnancies and a higher risk of complications. The patient's background of inadequate antenatal care further compounds her risk of eclampsia. Regular prenatal checkups are essential for monitoring blood pressure, proteinuria, and other signs of preeclampsia. Early detection and intervention can significantly reduce the risk of eclampsia and its associated complications. However, in this case, the patient's limited access to healthcare and lack of consistent prenatal care likely hindered the early identification and management of preeclampsia, ultimately leading to the development of eclampsia.^{10,11}

The combination of the patient's young age, physiological immaturity, potential underlying risk factors, and inadequate antenatal care created a perfect storm for the development of eclampsia. This case underscores the importance of addressing the unique challenges faced by adolescent pregnancies, including improving access to healthcare, providing comprehensive prenatal care, and educating teenagers about the risks and complications of pregnancy. Early identification and management of preeclampsia in adolescents are crucial for preventing the progression of eclampsia and improving maternal and fetal outcomes. Furthermore, this case highlights the need for further research into the pathophysiology of eclampsia in adolescents. Understanding the specific mechanisms underlying the increased risk in this population can lead to the development of targeted interventions and preventive strategies. Additionally, research is needed to identify optimal treatment approaches for eclampsia in adolescents, considering their unique physiological and developmental characteristics.12,13

The patient's limited access to healthcare and inadequate antenatal care are pivotal factors that likely contributed to the delayed diagnosis of atypical eclampsia in this case. This 16-year-old primigravida, hailing from a rural and low-income background, faced significant barriers to accessing comprehensive healthcare services. These barriers are multifaceted and often deeply ingrained in socioeconomic disparities, geographical constraints, and cultural beliefs. In many rural and low-income settings, healthcare facilities may be scarce, understaffed, and ill-equipped to handle complex obstetric cases like preeclampsia and eclampsia. Transportation to distant healthcare centers can be expensive and logistically challenging, further hindering access to care. Additionally, cultural beliefs and practices may discourage women from seeking prenatal care, especially in communities where traditional birth attendants are preferred over formal healthcare providers. The consequences of limited healthcare access and inadequate antenatal care are far-reaching and can have devastating impacts on maternal and fetal health. Regular prenatal checkups are essential for monitoring the progression of pregnancy, identifying potential complications, and initiating timely interventions. In the context of preeclampsia and eclampsia, these checkups are crucial for detecting early warning signs such as elevated blood pressure and proteinuria.13,14

Early detection of preeclampsia allows for prompt initiation of appropriate management strategies, such as antihypertensive therapy, magnesium sulfate for seizure prophylaxis, and close monitoring of maternal and fetal well-being. These interventions can significantly reduce the risk of progression to eclampsia, a life-threatening condition characterized by seizures and multi-organ dysfunction. In this particular case, the patient's lack of consistent prenatal care meant that her preeclampsia likely went undetected and untreated. The absence of regular blood pressure monitoring and urine testing masked the insidious development of the disease. As a result, the patient presented with sudden and recurrent seizures as the initial manifestation of eclampsia, a scenario associated with increased maternal and perinatal morbidity and mortality. The delayed diagnosis of atypical eclampsia in this patient underscores the critical importance of addressing healthcare disparities and improving access to antenatal care, especially in vulnerable populations. This requires a multi-pronged approach, including strengthening healthcare infrastructure in rural and underserved areas, increasing awareness about the importance of prenatal care, and addressing cultural barriers that may hinder healthcare-seeking behavior. Furthermore, healthcare providers should be vigilant in identifying and managing preeclampsia, even in the absence of classic symptoms. Atypical eclampsia, as seen in this case, can present without the typical signs of hypertension and significant proteinuria, making diagnosis more challenging. A high index of suspicion, thorough clinical assessment, and appropriate laboratory investigations are essential for early detection and timely intervention.^{14,15}

Magnesium sulfate (MgSO4) has long been considered the cornerstone of eclampsia treatment due to its well-established efficacy in preventing and controlling seizures. It acts as a central nervous system depressant, reducing neuronal excitability and stabilizing the electrical activity of the brain. Additionally, magnesium sulfate has been shown to have vasodilatory effects, improving blood flow to vital organs and potentially mitigating the end-organ damage associated with eclampsia. Numerous clinical trials meta-analyses have consistently and demonstrated the superiority of magnesium sulfate over other anticonvulsants, such as phenytoin and diazepam, in preventing recurrent seizures and improving maternal and fetal outcomes in eclampsia. Despite its widespread use and proven efficacy, a subset of patients with eclampsia may experience refractory seizures that do not respond to magnesium sulfate therapy. This phenomenon, known as magnesium-resistant eclampsia, poses a significant challenge in clinical practice. Several factors may contribute to magnesium resistance, including genetic variations in magnesium transporters, altered magnesium metabolism during pregnancy, and the underlying pathophysiology of eclampsia itself. In patients with magnesium-resistant eclampsia, alternative therapeutic agents are necessary to control seizures and prevent further complications. Benzodiazepines, such as midazolam and diazepam, are often used as second-line agents due to their rapid onset of action and potent anticonvulsant effects. However, benzodiazepines may cause respiratory depression and hypotension, particularly in patients with compromised cardiorespiratory function.^{15,16}

In recent years, dexmedetomidine, an alpha-2 adrenergic agonist, has emerged as a promising alternative for managing refractory seizures in eclampsia. Dexmedetomidine acts on the alpha-2 adrenergic receptors in the locus coeruleus, a brainstem region involved in arousal and stress response. By inhibiting norepinephrine release, dexmedetomidine produces sedation, analgesia, and anxiolysis. Additionally, dexmedetomidine has been shown to have neuroprotective effects, reducing neuronal damage and inflammation in various neurological conditions. Several studies have investigated the use of dexmedetomidine in eclampsia, with promising results. A randomized controlled trial comparing dexmedetomidine with magnesium sulfate for the prevention of shivering during spinal anesthesia for cesarean section found that dexmedetomidine was more effective in preventing shivering and had fewer side effects. Another study reported a case series of five women with eclampsia who were successfully treated with dexmedetomidine as an adjuvant to magnesium sulfate. The authors concluded that dexmedetomidine may be a safe and effective option for managing refractory seizures in The mechanisms eclampsia. underlying dexmedetomidine's anticonvulsant effects are not fully understood, but several hypotheses have been Dexmedetomidine proposed. mav modulate neurotransmitter release, inhibit calcium influx into neurons, and reduce the production of proinflammatory cytokines. These actions may contribute to its neuroprotective and anticonvulsant effects in eclampsia. The use of dexmedetomidine in eclampsia is not without potential risks and limitations. Dexmedetomidine can cause bradycardia and hypotension, which may be detrimental in patients with pre-existing cardiovascular compromise. Additionally, the long-term effects of dexmedetomidine on maternal and fetal outcomes are not wellestablished. Therefore, further research is needed to determine the optimal dose, duration, and safety profile of dexmedetomidine in eclampsia.^{16,17}

The successful utilization of midazolam and dexmedetomidine in this case of refractory eclampsia underscores their potential as valuable additions to the therapeutic armamentarium for managing this severe pregnancy complication. Both medications offer unique pharmacological properties that contribute to their efficacy in controlling seizures and mitigating potential neurological damage. Midazolam, a shortacting benzodiazepine, is renowned for its rapid onset of action and potent anticonvulsant effects. It acts by enhancing the inhibitory effects of gammaaminobutyric acid (GABA), a neurotransmitter that reduces neuronal excitability. By binding to specific GABA receptors, midazolam increases the frequency of chloride channel opening, leading to hyperpolarization of neurons and suppression of seizure activity. This rapid and effective mechanism of action makes midazolam an ideal choice for terminating acute seizures, particularly in emergency situations like eclampsia, where prompt seizure control is crucial to prevent maternal and fetal morbidity. In addition to its anticonvulsant properties, midazolam also possesses sedative effects, which can be beneficial in managing agitated or anxious patients. Sedation can help reduce the metabolic demands associated with seizure facilitate activity and patient management, particularly in the intensive care unit (ICU) setting. However, it is important to note that midazolam's sedative effects can also lead to respiratory depression, especially in patients with compromised respiratory function. Therefore, close monitoring of respiratory status is essential when administering midazolam, particularly in critically ill patients.17,18

Dexmedetomidine, an alpha-2 adrenergic agonist, offers a distinct pharmacological profile with both neuroprotective and anticonvulsant properties. It acts by binding to alpha-2 adrenergic receptors in the central nervous system, leading to a decrease in sympathetic outflow and an increase in parasympathetic activity. This results in a reduction in heart rate, blood pressure, and overall stress response, which can be beneficial in eclampsia patients who may experience hemodynamic instability and increased sympathetic activity due to seizures. The neuroprotective effects of dexmedetomidine are attributed to its ability to reduce cerebral blood flow and metabolism, thereby decreasing oxygen demand and mitigating potential ischemic injury. Additionally, dexmedetomidine has been shown to inhibit the release of excitatory neurotransmitters, such as glutamate, and enhance the release of inhibitory neurotransmitters, such GABA. as further contributing to its anticonvulsant effects. These neuroprotective and anticonvulsant properties make dexmedetomidine a promising agent for managing seizures in various neurological conditions, including eclampsia. The combination of midazolam and dexmedetomidine may offer synergistic effects in controlling seizures and providing sedation in eclampsia. Midazolam's rapid onset of action and anticonvulsant properties can potent quickly terminate seizures, while dexmedetomidine's longer duration of action and neuroprotective effects can help prevent seizure recurrence and mitigate potential neurological damage. Furthermore, the sedative properties of both medications can facilitate patient management and reduce the need for additional sedatives, which may have adverse effects on maternal and fetal outcomes.18,19

The use of midazolam and dexmedetomidine in this case of refractory eclampsia is consistent with emerging evidence suggesting their potential benefits in this context. Several studies have reported successful seizure control and improved maternal and fetal outcomes with the use of these agents in eclampsia patients who failed to respond to magnesium sulfate therapy. However, further research is needed to establish the optimal dosing regimens, safety profiles, and long-term outcomes associated with the use of midazolam and dexmedetomidine in eclampsia. The successful use of midazolam and dexmedetomidine in this case of refractory eclampsia highlights their potential as valuable additions to the therapeutic armamentarium for managing this severe complication. Their pregnancy unique pharmacological properties, including rapid onset of action, potent anticonvulsant effects, neuroprotection, and sedation, make them promising agents for controlling seizures and mitigating potential neurological damage in eclampsia. However, further research is needed to validate these findings and establish optimal treatment protocols for their use in this context. The combination of midazolam and dexmedetomidine may offer synergistic effects in controlling seizures and providing sedation in eclampsia. Midazolam's rapid onset of action and potent anticonvulsant properties can quickly terminate seizures, while dexmedetomidine's longer duration of action and neuroprotective effects can help prevent seizure recurrence and mitigate potential neurological damage. Additionally, dexmedetomidine's sedative properties can facilitate patient management and reduce the need for additional sedatives, which may have adverse effects on maternal and fetal outcomes. The patient's response to midazolam and dexmedetomidine underscores the importance of individualized treatment approaches in eclampsia. While magnesium sulfate remains the first-line treatment, alternative agents like midazolam and dexmedetomidine may be considered in cases of refractory seizures. The choice of antiepileptic agent should be based on the patient's clinical condition, seizure severity, and potential drug interactions.^{16,18}

The patient's postpartum course was marked by persistent and recurrent seizures, a complication that necessitated ongoing intensive care management. This underscored the severity of her atypical eclampsia and the challenges in achieving seizure control. The persistence of seizures postpartum, despite initial treatment with magnesium sulfate, highlighted the refractory nature of her condition and the need for additional therapeutic interventions. The decision to administer prophylactic antibiotics, specifically ceftriaxone, was a standard practice in the management of eclampsia patients. This practice stems from the recognition that eclampsia is associated with a heightened risk of infection. The underlying mechanism for this increased susceptibility lies in the systemic inflammatory response triggered by eclampsia. This systemic inflammation can disrupt immune function, impairing the body's ability to fight off pathogens and increasing the likelihood of developing infections. In the context of eclampsia, the systemic inflammatory response is characterized by the release of pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNFa), interleukin-6 (IL-6), and interleukin-1 beta (IL-1 β). These cytokines play a crucial role in the pathogenesis of eclampsia, contributing to endothelial dysfunction, vasoconstriction, and organ damage. However, their excessive release can also have detrimental effects on the immune system.^{17,19}

Pro-inflammatory cytokines can suppress the activity of immune cells, such as neutrophils and macrophages, which are essential for fighting off infections. They can also disrupt the integrity of epithelial barriers, making it easier for pathogens to invade the body. Additionally, the systemic inflammatory response can lead to a hypercoagulable state, increasing the risk of thrombosis and further compromising tissue perfusion, which can create an environment conducive to infection. The use of prophylactic antibiotics in eclampsia patients aims to mitigate this increased risk of infection. Ceftriaxone, a broad-spectrum cephalosporin antibiotic, is often chosen due to its effectiveness against a wide range of bacteria, including those commonly implicated in postpartum infections. By administering ceftriaxone, clinicians aim to prevent the development of infections that could further complicate the patient's recovery and potentially lead to life-threatening sepsis. The administration of ceftriaxone in this case was not only a precautionary measure but also a strategic intervention to protect the patient from potential infectious complications. The patient's compromised immune status, resulting from the systemic inflammatory response associated with eclampsia, made her particularly vulnerable to infections. The use of prophylactic antibiotics aimed to safeguard her health and facilitate a smoother recovery process.^{15,18}

In addition to prophylactic antibiotics, the patient's intensive care management involved close monitoring of her vital signs, neurological status, and laboratory parameters. This comprehensive approach ensured that any signs of infection or other complications were promptly detected and addressed. The multidisciplinary team, consisting of obstetricians, anesthesiologists, intensivists, and neonatologists, worked collaboratively to provide the patient with the best possible care. The patient's eventual recovery and discharge from the hospital without any infectious underscore complications the importance of prophylactic antibiotics in the management of eclampsia. This case highlights the critical role of a multidisciplinary team in providing comprehensive care to eclampsia patients, addressing not only the immediate threat of seizures but also the potential long-term complications associated with this complex condition. The patient's ICU stay was characterized by close monitoring of vital signs, neurological status, and laboratory parameters. The multidisciplinary team, consisting of obstetricians, anesthesiologists, intensivists, and neonatologists, played a crucial role in ensuring the patient's recovery. The coordinated efforts of this team ensured that the patient received comprehensive care, addressing not only the seizures but also the potential complications of eclampsia, such as organ dysfunction and coagulopathy.16,19

The patient's eventual recovery and discharge further complications without highlight the importance of timely and appropriate intervention in eclampsia. Early recognition of atypical eclampsia, prompt initiation of antiepileptic therapy, and comprehensive supportive care are essential for improving maternal and perinatal outcomes. This case also emphasizes the need for further research to elucidate the pathophysiology of atypical eclampsia and to identify optimal treatment strategies for this challenging condition. This case report presents a unique case of atypical eclampsia in a young primigravida with recurrent seizures. The successful

management of this case with a combination of midazolam and dexmedetomidine suggests their potential as alternative therapeutic agents for refractory eclampsia. This case also underscores the importance of considering atypical eclampsia in pregnant women presenting with seizures, even in the absence of classic preeclampsia symptoms. Further research is needed to validate these findings and establish optimal treatment protocols for atypical eclampsia.²⁰

4. Conclusion

This case report demonstrates the successful management of recurrent seizures in a young primigravida with atypical eclampsia using a combination of midazolam and dexmedetomidine. The findings suggest that these agents may be considered alternative therapeutic options for refractory seizures in eclampsia. However, further research is needed to validate these findings and establish optimal treatment protocols for atypical eclampsia.

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