

Transient Anisocoria and Sudden Decrease in Consciousness Following Thoracic Epidural Analgesia: A Case Report

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Case Report

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Abstract

Background: Anisocoria during neuraxial anesthesia is an uncommon finding that may represent transient autonomic dysfunction or serious neurological complications, posing a diagnostic challenge for anesthesiologists.

Case Presentation: A previously healthy 33-year-old woman underwent elective breast implant exchange and liposuction with planned thoracic epidural analgesia combined with general anesthesia. Shortly after administration of 4 mL of an epidural solution containing levobupivacaine and morphine, she developed abrupt alteration of consciousness and anisocoria, characterized by left-sided mydriasis and right-sided miosis. Despite the neurological event, she remained hemodynamically stable and showed no signs of seizures, respiratory compromise, or cardiovascular collapse. Airway protection was promptly achieved with tracheal intubation and controlled ventilation. Serial pupillary and cardiorespiratory monitoring demonstrated complete spontaneous resolution of anisocoria approximately 60 minutes later. The postoperative course was uneventful, with no neurological deficits or recurrence of symptoms during 36 hours of observation.

Conclusion: Transient anisocoria associated with altered consciousness following epidural analgesia is rare and may be related to extensive cephalad neuraxial spread and transient sympathetic dysfunction. Early recognition, airway protection when indicated, and close neurological monitoring are essential for favorable outcomes.

INTRODUCTION

Anisocoria identified during anesthesia is an uncommon but clinically significant finding that may raise concern for acute neurological or neuro-ophthalmological pathology. In the perioperative setting, its interpretation can be particularly challenging because anesthetic drugs, neuraxial techniques, autonomic blockade, and changes in intracranial or ocular physiology may produce transient pupillary abnormalities that can mimic more serious central neurological conditions. Prompt recognition and systematic assessment are therefore essential to distinguish potentially life-threatening causes from reversible anesthesia-related phenomena. [1, 2]

Epidural analgesia is an established component of multimodal perioperative analgesia, providing effective pain control while reducing perioperative opioid requirements. Although generally considered safe, neuraxial administration of local anesthetics and adjuvant drugs may occasionally result in unexpected neurological manifestations. These effects may arise from unintended intravascular or intrathecal administration, extensive cephalad spread, inadvertent subdural spread, or blockade of autonomic pathways. Transient pupillary abnormalities following epidural analgesia are uncommon and may present a diagnostic challenge, particularly when accompanied by an acute alteration in consciousness. [3, 4]

Transient Horner's syndrome has also been described as a rare complication of neuraxial anesthesia, including thoracic and lumbar epidural techniques. It is thought to result from cephalad spread of local anesthetic to the upper thoracic and cervical sympathetic pathways, potentially producing ipsilateral miosis, ptosis, anhidrosis, and facial flushing. Although usually self-limited, the occurrence of pupillary asymmetry in the perioperative setting may be difficult to distinguish from other neurological conditions and therefore warrants careful clinical assessment and monitoring. [5, 6, 7] The coexistence of altered consciousness and anisocoria following epidural administration is particularly unusual and may reflect extensive neuraxial spread involving both autonomic and central nervous system functions. [8]

We report the case of a previously healthy 33-year-old woman who developed abrupt alteration in consciousness and transient anisocoria following the initial administration of thoracic epidural analgesia during elective surgery. The absence of significant cardiovascular or respiratory compromise, together with the spontaneous resolution of the pupillary abnormality and the absence of postoperative neurological sequelae, illustrates the diagnostic uncertainty and perioperative management challenges associated with acute neurological findings during neuraxial anesthesia. This case highlights the importance of prompt airway assessment and protection when indicated, continuous cardiorespiratory monitoring, serial neurological and pupillary assessment, and careful reassessment before emergence and extubation.

CASE REPORT

A 33-year-old woman, classified as American Society of Anesthesiologists (ASA) physical status I and considered previously healthy, was admitted for elective breast implant exchange combined with liposuction. She had no significant medical history, including the absence of known comorbidities, chronic medication use, or documented drug allergies. Her surgical background included cesarean delivery five years earlier and breast augmentation, both performed without anesthetic or surgical complications.

During the pre-anesthetic evaluation, the patient weighed 62 kg and was 1.62 m tall, corresponding to a body mass index (BMI) of 23.6 kg/m². Physical examination revealed no relevant abnormalities, and laboratory and complementary examinations were within normal limits. Airway assessment demonstrated favorable predictors for tracheal intubation, including adequate mouth opening, Mallampati class I, thyromental distance greater than 3 cm, preserved atlanto-occipital mobility, and absence of clinical predictors of difficult airway management.

Considering the surgical procedure and the expected postoperative analgesic requirements, a combined anesthetic approach consisting of general anesthesia associated with thoracic epidural analgesia was planned, aiming to optimize perioperative pain control and reduce postoperative opioid consumption. Upon arrival in the operating room, standard monitoring was established according to ASA recommendations, including continuous electrocardiography, pulse oximetry, and non-invasive blood pressure monitoring. Baseline parameters were as follows: blood pressure 132/85 mmHg, heart rate 78

beats/min, peripheral oxygen saturation 99% on room air, and Ramsay sedation score II. A large-bore peripheral intravenous catheter (18G) was inserted, followed by conscious sedation with intravenous midazolam (3 mg) and fentanyl (20 µg).

During this period, the patient maintained spontaneous ventilation, hemodynamic stability, and adequate responsiveness to verbal commands. With the patient in the sitting position, thoracic epidural catheterization was performed at the T11–T12 interspace using an 18-gauge Tuohy needle and the loss-of-resistance technique. The epidural space was identified unequivocally on the first attempt. After negative aspiration for blood and cerebrospinal fluid, a 3-mL test dose of 2% lidocaine with epinephrine (1:200,000) was administered while the patient remained in the sitting position. No clinically significant increase in heart rate or other hemodynamic changes suggestive of intravascular injection were observed.

The epidural catheter was subsequently advanced without resistance, and the patient reported no pain, paresthesia, or other neurological symptoms during its progression. The catheter was then secured while the patient remained in the sitting position. Following another negative aspiration test, incremental administration of the epidural analgesic solution was initiated.

Following another negative aspiration test, incremental administration of the epidural analgesic solution was initiated. The solution consisted of 14 mL of 0.5% levobupivacaine (70 mg), 1 mg of morphine in 1 mL, and 5 mL of distilled water, resulting in a total volume of 20 mL. An initial volume of 4 mL was administered while the patient remained in the sitting position.

Following administration of 4 mL of the epidural solution, the patient was placed in the supine position. Shortly thereafter, an abrupt reduction in the level of consciousness was observed, accompanied by anisocoria, characterized by left-sided mydriasis and right-sided miosis. The patient remained hemodynamically stable, with a heart rate of approximately 75 beats/min, preserved blood pressure, and adequate peripheral perfusion. There were no episodes of significant bradycardia, hypotension, relevant oxygen desaturation, seizures, tonic-clonic movements, or muscular rigidity.

Given the acute neurological presentation and the possibility of extensive cephalad neuraxial spread, airway protection was promptly undertaken. Intravenous etilefrine (3 mg) was administered, followed by induction of general anesthesia with propofol (150 mg), fentanyl (100 µg), and cisatracurium (10 mg). Tracheal intubation was successfully performed on the first attempt under direct laryngoscopy, without difficulty, using a 7.0-mm endotracheal tube. Correct tracheal tube placement was confirmed by capnography, and controlled mechanical ventilation was initiated.

Following tracheal intubation and maintenance of anesthesia, continuous cardiorespiratory monitoring was maintained, including electrocardiography, pulse oximetry, non-invasive blood pressure measurements, and continuous capnographic assessment. Mechanical ventilation was instituted with close observation of oxygenation and ventilation. Given the acute neurological event, serial pupillary assessments were performed throughout the intraoperative period, along with continuous surveillance

for hemodynamic instability, changes in ventilatory parameters, recurrent neurological abnormalities, or other signs suggestive of progressive neuraxial effects or systemic local anesthetic toxicity.

No significant hypotension, bradycardia, arrhythmia, oxygen desaturation, or ventilatory instability occurred after airway management. No additional vasopressor doses were required. The surgical procedure was therefore continued under general anesthesia with TIVA and close clinical monitoring. Approximately 60 minutes after the beginning of the surgical procedure, complete spontaneous resolution of the anisocoria was observed, with restoration of bilateral pupillary symmetry. No further neurological abnormalities were identified during the remainder of the procedure. The surgical procedure lasted approximately four hours and proceeded without further anesthetic or surgical complications.

At the end of surgery, the patient was reassessed before extubation. She demonstrated adequate spontaneous ventilation, adequate neuromuscular recovery, appropriate response to verbal commands, stable hemodynamic parameters, and no clinically evident neurological abnormalities. Given the complete resolution of the anisocoria and the absence of residual signs suggestive of neurological deterioration or persistent neuraxial effects, the patient was considered suitable for safe extubation.

Following extubation, the patient remained in the operating room for 30 minutes for continued clinical observation before transfer to the post-anesthesia care unit (PACU). Continuous monitoring was maintained in the PACU for approximately four hours, with serial assessment of level of consciousness, pupillary symmetry, motor and sensory function, respiratory status, and hemodynamic stability.

Given the unusual intraoperative neurological event, prolonged postoperative observation was elected. The patient subsequently remained hospitalized for approximately 36 hours. Throughout this period, she remained fully oriented and neurologically intact, without recurrent anisocoria, headache, visual disturbances, motor or sensory deficits, or other focal neurological findings. She was discharged home in good clinical condition, without apparent neurological sequelae.

The patient was informed about the unusual perioperative event and its clinical implications. At follow-up, she reported satisfaction with the care provided and expressed relief regarding the complete neurological recovery without residual symptoms.

DISCUSSION

The present case illustrates an uncommon and diagnostically challenging neurological event occurring immediately after the initial administration of thoracic epidural analgesia. The combination of abrupt alteration in consciousness and anisocoria in a previously healthy patient, in the absence of significant cardiovascular or respiratory instability, raised several potential differential diagnoses, including extensive cephalad neuraxial spread, inadvertent subdural or intrathecal spread, transient sympathetic blockade, local anesthetic systemic toxicity (LAST), and an acute central neurological event.

The temporal relationship between epidural administration and symptom onset, together with subsequent spontaneous resolution, favors a transient neuraxial mechanism. Cephalad spread of epidurally administered local anesthetic may reach upper thoracic and cervical sympathetic pathways and has been associated with transient Horner's syndrome after epidural anesthesia. The oculosympathetic pathway is particularly susceptible to high neuraxial blockade, and its transient interruption may produce pupillary asymmetry and other manifestations of sympathetic dysfunction. Previous reports have described Horner's syndrome as an uncommon but generally reversible complication of epidural anesthesia, emphasizing the importance of close observation for associated autonomic effects. [3–7]

In the present case, however, the pupillary abnormality consisted of left-sided mydriasis and right-sided miosis, without documented ptosis, anhidrosis, or other classical clinical features sufficient to establish Horner's syndrome. Therefore, it would be inappropriate to classify the event definitively as Horner's syndrome. Instead, the anisocoria may represent transient autonomic dysfunction associated with cephalad neuraxial spread. The complete spontaneous resolution of the pupillary asymmetry approximately 60 minutes after the beginning of surgery further supports a reversible pharmacological or neuraxial mechanism. The transient nature of similar epidural-associated sympathetic disturbances has been previously described. [3–7]

The abrupt alteration in consciousness represents an additional and particularly relevant aspect of this case. Extensive neuraxial blockade may produce neurological manifestations beyond the expected sensory distribution of an epidural technique. A previous case report described transient profound impairment of consciousness following epidural anesthesia, with complete neurological recovery and no permanent sequelae, supporting the possibility that extensive neuraxial effects may, in rare circumstances, be associated with marked alterations in alertness.⁸ In the present patient, the absence of significant hypotension, bradycardia, respiratory compromise, or persistent motor or sensory deficits makes a clinically severe total spinal block less evident. Nevertheless, the close temporal relationship between epidural administration and the neurological alteration, followed by complete recovery, makes extensive cephalad neuraxial spread an important consideration.

An inadvertent subdural injection also remains a plausible alternative explanation. Subdural spread may produce an atypical or disproportionately extensive neuraxial block despite apparently uncomplicated identification of the epidural space. Cases of Horner's syndrome and other neurological manifestations following epidural analgesia have been attributed to possible subdural spread, highlighting the difficulty of distinguishing this mechanism clinically from extensive epidural cephalad spread. However, in the present case, there was no direct evidence confirming subdural or intrathecal catheter placement. Therefore, this mechanism remains speculative. [7, 8, 9]

Local anesthetic systemic toxicity was also considered because levobupivacaine had been administered. However, the clinical presentation was not typical of severe LAST. The patient remained hemodynamically stable and did not develop seizures, significant arrhythmias, marked hypotension, or

cardiovascular collapse. Contemporary recommendations emphasize that LAST is a potentially life-threatening complication requiring prompt recognition and appropriate airway, cardiovascular, and resuscitative management when clinically suspected. Although the absence of these findings does not completely exclude LAST, the clinical evolution in this patient makes systemic toxicity less likely than a transient neuraxial or autonomic phenomenon. [10]

An acute structural neurological event was another important consideration at the time of presentation. Acute anisocoria may represent serious intracranial or neuro-ophthalmological pathology, and its temporal association with neuraxial drug administration should not, by itself, establish causality. In this patient, however, the absence of progressive neurological deterioration, persistent pupillary abnormality, focal neurological deficits, hemodynamic or respiratory instability, and postoperative recurrence, together with complete spontaneous resolution of the anisocoria, progressively reduced the likelihood of a persistent structural neurological lesion. Nevertheless, because neuroimaging and formal neurological assessment were not performed during the intraoperative event, a structural central cause cannot be considered definitively excluded. This limitation should therefore be acknowledged when interpreting the presumed mechanism.

The perioperative management was guided primarily by the acute neurological change and the potential for progression of neuraxial blockade. Rather than waiting for respiratory or cardiovascular compromise to develop, the team proceeded with early airway protection through tracheal intubation and controlled mechanical ventilation. This allowed adequate oxygenation and ventilation while providing physiological stability during continued observation of the neurological findings. Continuous cardiorespiratory monitoring and serial pupillary assessments were maintained throughout the procedure, together with surveillance for hemodynamic instability, ventilatory deterioration, recurrent neurological abnormalities, or other manifestations suggestive of progression of neuraxial blockade or systemic toxicity.

Importantly, no significant hypotension, bradycardia, arrhythmia, oxygen desaturation, or ventilatory instability occurred after airway management, and no additional vasopressor doses were required. The surgical procedure was therefore continued under general anesthesia with total intravenous anesthesia and close clinical monitoring. The decision to proceed was based on sustained physiological stability and the absence of progressive neurological deterioration rather than on the assumption that the initial event was benign.

The spontaneous resolution of anisocoria approximately 60 minutes after the beginning of surgery provided an important clinical indicator of reversibility. At the end of the procedure, extubation was performed only after recovery of spontaneous ventilation, neuromuscular function, consciousness, and clinically normal neurological status. Postoperative surveillance was deliberately extended, with approximately four hours of monitoring in the post-anesthesia care unit followed by approximately 36 hours of inpatient observation. During this period, the patient remained fully oriented and neurologically intact, with no recurrent anisocoria, visual symptoms, headache, motor or sensory deficits, or other focal neurological abnormalities.

From a practical perspective, this case emphasizes that anisocoria occurring after neuraxial anesthesia should not immediately be attributed to an isolated benign autonomic phenomenon. The initial response should prioritize airway and respiratory assessment, cardiovascular stabilization, consideration of high or extensive neuraxial blockade, and repeated neurological and pupillary examination. The decision to continue surgery should be individualized according to the patient's physiological stability and the evolution of neurological findings. Similarly, extubation should be considered only after adequate recovery of consciousness, ventilation, neuromuscular function, and neurological status.

The absence of definitive diagnostic testing represents an important limitation of this report. Neuroimaging or formal neurological evaluation during the event might have provided additional information regarding the possibility of a structural central lesion or other neuro-ophthalmological causes of anisocoria. Likewise, without direct evidence of intrathecal or subdural catheter placement, the exact anatomical mechanism cannot be established. Nevertheless, the close temporal relationship between epidural administration and symptom onset, the absence of manifestations strongly suggestive of severe LAST or cardiovascular collapse, the spontaneous resolution of anisocoria, and the complete neurological recovery during prolonged postoperative observation collectively support a transient neuraxial or autonomic mechanism as the most plausible explanation.

This case reinforces the importance of maintaining a broad differential diagnosis when acute neurological signs occur during neuraxial anesthesia. Early recognition, prompt airway protection when clinically indicated, continuous physiological monitoring, serial pupillary and neurological assessment, and cautious reassessment before extubation may contribute to favorable outcomes when the underlying process is transient and reversible.

Strengths and limitations of this case report

The main limitation is the inability to establish a definitive causal relationship between epidural analgesia and the neurological findings due to the inherent nature of a single case report. Additionally, the absence of advanced neurological imaging or specific diagnostic testing limits further investigation of the underlying mechanism.

The main strength of this case report is the detailed description of an uncommon perioperative neurological event, including the clinical presentation, anesthetic management, and complete neurological recovery. The case provides valuable insights into the recognition and management of transient anisocoria following neuraxial anesthesia in an otherwise healthy patient.

Declarations

Ethics Approval and Consent to Participate

Ethics committee approval was not required for this case report, in accordance with the applicable institutional and local regulations for the publication of individual case reports. The case report was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical information.

Consent to Publish: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available if needed for review by the Editor-in-Chief of this journal.

Availability of Data and Materials

The data supporting the findings of this case report are available from the corresponding author upon reasonable request, subject to appropriate ethical and privacy considerations.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

PSB contributed to the conception and design of the case report, clinical management, data collection, interpretation of the clinical findings, and drafting and revision of the manuscript.

ABOJ contributed to the clinical management, data collection, interpretation of the clinical findings, and critical revision of the manuscript.

NSPM contributed to the conception of the report, clinical interpretation, and critical revision of the manuscript.

SLG contributed to the clinical management, data collection, interpretation of the findings, and critical revision of the manuscript.

MYG contributed to the clinical management, interpretation of the findings, and critical revision of the manuscript.

EBB contributed to the literature review, interpretation of the clinical findings, and critical revision of the manuscript.

All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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