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Anisocoria and mydriasis after scalp nerve block: a case report

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Abstract

Strategies for the assessment of abnormal neurological findings during general anesthesia are limited. However, pupil abnormalities may represent serious neurological complications. We herein present a case of new-onset anisocoria and mydriasis that developed after scalp nerve block. The patient's signs were possibly related to increased intracranial pressure with resulting brain shift that ultimately affected the oculomotor nerves. A 45-year-old man was scheduled for left cerebellar tumor resection and ventricular drainage surgery; however, anisocoria and left pupillary mydriasis were observed after induction of general anesthesia and performance of scalp nerve block. After reducing the intracranial pressure, the right pupil showed constriction (1 mm) but the left pupil was dilated (5 mm). The pupils were of similar size postoperatively. Although pupillary dilation during general anesthesia has been previously described, this is the first case in which the mydriasis was considered to have been caused by brain shift due to increased intracranial pressure after scalp nerve block. Thus, we propose this phenomenon as a new possible cause of pupillary changes. Actively monitoring this presentation intraoperatively could enable early detection of and intervention for complications, therefore improving the prognosis.

Keywords

Anisocoria, brain shift, increased intracranial pressure, mydriasis, scalp nerve block, case report

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Introduction

Anisocoria and mydriasis are significant neurological findings that can indicate underlying life-threatening conditions. Perioperative examination of the pupil size is of great importance for anesthesiologists to understand the patient's present illness,

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medical history, and use of drugs; this examination is especially important for patients scheduled for neurosurgery. Physiological, physical, and pathological factors can affect the pupil size, and early identification of the factor(s) causing pupil changes is crucial. This enables judgment of whether the patient requires urgent diagnosis to exclude serious pathology, thereby improving the prognosis. In this report, we describe a case of new-onset anisocoria and mydriasis that developed after scalp nerve block. The findings in this case suggest that the procedures involved in scalp nerve block may cause pupillary changes secondary to increased intracranial pressure (ICP). Anesthesiologists are often the first to discover bilateral pupillary changes intraoperatively; therefore, careful differential diagnosis is required to ensure the patient's safety and avoid excessive medical treatment.

Case presentation

A 45-year-old man underwent surgery in 2017 for a cerebellar tumor that had been causing headaches. In June 2020, the headaches recurred with additional symptoms of intermittent pain in the parieto-occipital region, vomiting after eating at night, and non-projectile vomiting. At presentation, the patient reported no fasciculations, blurred vision, or diplopia. Besides the cerebellar tumor in 2017, he had no history of any other disease. Physical examination revealed no abnormalities; his vital signs were within normal limits, and his consciousness was clear. His pupils were equally sized and approximately 2 mm in diameter, and they exhibited a normal light reflex. His neck was soft, and the muscle strength in his limbs was normal. Brain magnetic resonance imaging indicated a left cerebellar hemispheric space-occupying lesion (3.8×4.8 cm), suggesting tumor recurrence. The fourth ventricle was compressed and narrowed, the midline structures were normal, and supratentorial hydrocephalus was present. All other laboratory data were within the acceptable ranges. The patient consented to left cerebellar tumor resection and ventricular drainage.

In the operating room, we examined the patient's pupils, which were approximately 2 mm in diameter. At 8:05 AM, general anesthesia was induced by intravenous injection of 4 mg midazolam, 6 mg lidocaine, 20 μg sufentanil, 70 mg propofol, and 60 mg rocuronium. Following intubation with a 7.0-mm cuffed tracheal tube, anesthesia was maintained with continuous infusion of propofol and remifentanil. The scalp nerve block was completed at 8:25 AM (a total of 20 mL of 0.25% ropivacaine was used; each greater occipital nerve was blocked with 3 mL, each lesser occipital nerve was blocked with 3 mL, the auriculotemporal nerve was blocked with 2 mL, and the supraorbital nerve was blocked with 2 mL), at which time the patient's heart rate rapidly increased from to 80 beats/minutes while his blood pressure remained constant. At 8:33 AM, the right pupil measured 4 mm in diameter, the left pupil measured 5 mm, the heart rate dropped to the rate observed before the nerve block, and the vital signs were stable. Considering the possibility of increased ICP, 250 mL of 20% intravenous mannitol was immediately administered. Arterial blood gas analysis showed no hypoxemia or carbon dioxide retention.

The pupils were examined again at 8:55 AM, at which time the right pupil was 3 mm and the left pupil was 5 mm. A repeat blood gas analysis showed no abnormalities. Twenty-five minutes later, 20 mg intravenous furosemide was administered, and examination with the O-arm surgical imaging system showed no supratentorial hemorrhage. Seven minutes later, the right pupil size was 1 mm and the left was 5 mm. At the

same time, we communicated with the patient's family and informed them in detail of the special condition of the patient after anesthesia. With the understanding and consent of his family members, we began the operation. At the end of the operation, both pupils were of equal size (diameter of 1 mm). The patient was transferred to the neurosurgical care unit, where a complete neurosurgical examination was performed. After the patient awoke, his pupils were still of equal size (diameter of 3 mm) with a normal reaction to light. The neurosurgical team performed brain computed tomography, and the results indicated no intracranial hemorrhage or ischemic findings; furthermore, an ophthalmology consultation revealed no abnormalities. The reporting of this case conforms to the CARE guidelines.[1]

Discussion

Although a $\geq$1-mm difference in pupil diameter is a physiological phenomenon observed in 20% of the general population,[2] acute anisocoria and mydriasis is a disturbing and unusual finding during the perioperative period. Because it may be caused by various life-threatening etiologies or exposure of the eyes to drugs, it is a great concern among anesthesiologists. Therefore, proper knowledge of the probable causes of abnormal anisocoria and mydriasis is immensely valuable for appropriate evaluation and timely management. Various drugs used during anesthesia can cause pupillary changes. Dexmedetomidine, neostigmine, morphine, drugs that induce hibernation-like effects, barbiturates, and phenothiazines can cause miosis. Atropine, epinephrine, norepinephrine, ephedrine, and scopolamine can induce mydriasis. Pupillary changes caused by systemic medication are usually bilateral and consistent.

Pupillary dilation is mediated by a balance between the sympathetic and parasympathetic pathways. Pupillary changes can also be caused by systemic factors (such as increased ICP due to neurological and vascular diseases and trauma or hypoxic injury of the autonomic nervous system) or local factors (such as direct damage to the eye during intubation or exposure to sympathetic or anticholinergic drugs in and around the unilateral eye and nose).[3,4] One study showed that pupillary dilation indicates diffusion of epinephrine through the nasolacrimal duct into the eye, causing unilateral pupillary dilation.[5] Prielipp[4] reported mydriasis following the application of phenylephrine/lidocaine spray to the airway for topical anesthesia to the airway. The author speculated that lidocaine may have entered the orbit through the pterygopalatine fossa and superior orbital fissure, thereby blocking the ciliary ganglion.[4]

Miosis is a common presentation after induction of general anesthesia. In the present case, however, the patient's pupils were dilated and of unequal size. The patient had no history of eye diseases, and his pupil sizes normalized after he regained consciousness postoperatively. Moreover, ocular disease was excluded by the postoperative ophthalmic consultation. Furthermore, we used no drugs intraoperatively that could have caused pupillary dilation, allowing us to exclude drugs as a potential etiology of the patient's mydriasis. The right pupil gradually constricted to 3 mm after mannitol administration and further to 1 mm after furosemide administration; however, the left pupil remained dilated. The O-arm examination excluded intracranial hemorrhage, and isocoria (diameter of 1 mm) was observed at the end of surgery.

After a comprehensive analysis, we considered that the pupillary changes in this patient might have been related to brain shift caused by increased ICP. Brain shift is a serious complication with a wide range of causes, among which ICP change

is a major concern. When the volume of intracranial structures increases to a certain extent, intracranial compliance is reduced; moreover, even a slight increase in the volume of intracranial structures can cause a significant increase in ICP, which may lead to brain shift or even cerebral herniation. Brain shift can also be caused by gravity, aggressive intraoperative dehydration, cerebrospinal fluid aspiration, surgical procedures, certain tumor types, anesthetics, and mechanical ventilation.[6,7]

Brain shift is a routine occurrence during deep brain stimulation neurosurgery.[8,9] As early as 1972, human and animal studies showed that an increase in ICP during the administration of anesthetic agents, such as halothane, is greatly exacerbated by the presence of pre-existing intracranial pathology, which reduces the capacity for volume compensation. Under these circumstances, cerebral perfusion pressure may be reduced, and exacerbation of pressure gradients within the cranial cavity may lead to brain shift; this can result in brain stem compression,[10,11] which can indirectly affect the pupillary size. Displacement of the cerebellar vermis through the tentorium is likely when the ICP is increased because of a cerebellar tumor, hemorrhage, or other causes, even when upward transtentorial herniation has occurred. The tentorial notch varies in length and width (44–75 mm and 26–30 mm, respectively). Upward herniation of the superior cerebellar vermis may easily occur when the tentorial opening is larger. As the upward herniation progresses, the shifted cerebellar vermis distorts the midbrain and cerebral aqueduct and buckles the quadrigeminal plate; this allows the inferior colliculi to fold under the superior colliculi, and together, both structures shift upward beneath the splenium of the corpus callosum.[12] At the level of the tentorial notch, the superior vermis of the cerebellum lies close to the dorsum of the midbrain. The midbrain is vulnerable to displacement by forces from below and above the tentorium. In a retrospective analysis of seven patients with upward transtentorial herniation, Cuneo et al.[13] highly suspected that the pupillary changes observed in the patient were related to the distortion and buckling of the midbrain and oculomotor nerves due to the upward transtentorial herniation. Therefore, we speculate that the changes in our patient's pupil were related to cerebellar tissue/tumor displacement caused by an increase in ICP, which ultimately affected the oculomotor nerves.

The specific mechanism underlying the ICP increase in this patient after anesthesia induction and scalp nerve block remains unclear; however, we considered that lateral head rotation likely resulted in the obstruction of venous drainage. Williams and Coyne[14] investigated the effect of head rotation to the left and right, neck flexion, and neck extension on ICP in 10 patients whose ICP was being therapeutically monitored but who were not experiencing prolonged periods of elevated ICP. The authors found that the highest ICP always occurred when the head and neck were rotated or in flexion. This result suggests that a patient at risk for a pathological increase in ICP should not bend the neck or turn the head to either side.[14] Mavrocordatos et al.[15] reported the effect of the positions of the operating table, head, and neck on ICP in 15 adult patients during anesthesia and demonstrated that ICP increased every time the head was in a non-neutral position. Using ultrasound color Doppler imaging, Burbridge et al.[16] demonstrated that mechanical ventilation under general anesthesia can occlude the left and right internal jugular veins at average lateral rotations of 55.6° and 53.3°, respectively, and that the ICP can increase substantially when the internal jugular vein is occluded. Hung et al.[17] found that head rotation of 60° maximally increased the ICP

to 24 ± 14.3 mmHg, whereas head elevation reduced the intracranial hypertension associated with head rotation. The head must be rotated from side to side while administering the bilateral greater and lesser occipital nerve blocks, which may increase ICP. At the same time, we observed a temporary increase in the patient's heart rate in the present case. Dimitri et al.[18] also found that in patients with traumatic brain injury, a temporary increase in ICP and heart rate can be observed before a severe increase in the mean ICP. Therefore, we suspect that changes in heart rate may be related to increases in ICP, which further supports our conjecture.

The phenomenon of anisocoria or mydriasis during anesthesia is not uncommon, although many potential benign factors may be responsible. However, there is limited scope for neurological function assessment because of the patient's anesthetized state. Moreover, an insufficient understanding of the causes of preoperative pupillary changes will cause doctors to focus excessive attention on the case and increase the clinical input needed during the perioperative period. In cases of vision-related and life-threatening neurological diseases, early detection of new pupillary changes can enable early intervention and treatment, improving the patient's prognosis. In this report, the most likely explanation is that head rotation during the scalp nerve block obstructed the venous drainage and increased the ICP, resulting in brain displacement and ultimately affecting the oculomotor nerve. We propose a new possible mechanism of pupillary changes during general anesthesia: anisocoria or mydriasis may be caused by increased ICP after scalp nerve block. Therefore, we strongly recommend that anesthesiologists perform simple pupil examinations as part of the routine preoperative anesthesia evaluation while also intermittently evaluating the pupil size before, during, and after the operation to facilitate the diagnosis of probable causes. In addition, several studies have confirmed that reducing the head rotation angle or raising the head to $\geq 30^{\circ}$ can effectively avoid increases in ICP.[16,17] Therefore, we strongly suggest that during scalp nerve block, the operating table should be adjusted to the high position of the head and excessive head rotation should be avoided, thereby preventing an ICP increase. This is particularly important for patients who require neurosurgery.

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Author contributions

XC and FC reviewed the literature and wrote the manuscript.
SJ and YT examined the patient and performed the anesthesia.
XB and SJ reviewed and edited the manuscript.
All authors read and approved the manuscript.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

Ethics approval and consent to participate

The requirement for ethics approval was waived because of the nature of this study (case report). The patient provided written informed consent for the publication of this case.

Data availability statement

The data and materials of the report are available from the corresponding author upon reasonable request.

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