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Case Report

Acute anisocoria secondary to increased intra-abdominal pressure: A case series and mini review of the literature

Asif Dabeer Jafri[1], *Ratender Kumar Singh*[2], *Tanmoy Ghatak*[2]

[1] *Department of Emergency Medicine and Tele-ICU Service, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India*
[1] *Department of Emergency Medicine, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India*

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ABSTRACT

Introduction: In the intensive care unit (ICU), acute-onset anisocoria is typically considered a sign of intracranial pathology with increased intracranial pressure (ICP), often resulting in third cranial nerve compression and necessitating urgent intervention. However, alternative etiologies such as elevated intra-abdominal pressure (IAP) in severe acute pancreatitis (SAP) can also precipitate increased ICP and anisocoria.

Aim: This report aims to examine the association between elevated IAP and the development of acute anisocoria with increased ICP in SAP patients and to evaluate the impact of early cerebroprotective measures on patient outcomes.

Case study: Three SAP patients in the ICU presented with sudden anisocoria. Despite initial concerns for central nervous system pathology, brain computed tomography (CT) scans were normal. Early cerebroprotective strategies were initiated in all cases. Two patients experienced complete resolution of anisocoria and stabilization of ICP, whereas one patient unfortunately succumbed to complications.

Results and discussion: These cases highlight that increased IAP can indirectly elevate ICP, manifesting as anisocoria in critically ill SAP patients. Clinicians should consider non-CNS etiologies when evaluating anisocoria, particularly when imaging findings are unremarkable. Non-invasive diagnostic tools, such as optic nerve sheath diameter measurement, may aid in the early detection of raised ICP and guide timely intervention.

Conclusions: Recognizing elevated IAP as a potential contributor to increased ICP and anisocoria is crucial for managing SAP patients. Early cerebroprotective interventions and timely intervention to lower IAP can improve outcomes, yet further research is needed to establish definitive IAP thresholds and optimize therapeutic strategies.

Corresponding author: Asif Dabeer Jafri, Department of Emergency Medicine and Tele-ICU Service, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India.
E-mail address: Asif_jafri2003@yahoo.co.in

1. INTRODUCTION

Anisocoria, defined as the presence of unequal pupil sizes, is often a significant observation in the intensive care unit (ICU) and is generally linked to elevated intracranial pressure (ICP) resulting from various neurological disorders, such as traumatic brain injury, stroke, and space-occupying lesions. It is essential to understand that anisocoria is not solely caused by central nervous system (CNS) pathology. Notably, acute anisocoria may also occur due to increased intra-abdominal pressure (IAP), which initiates a series of physiological responses that can lead to heightened ICP. This phenomenon is frequently overlooked in clinical settings. Severe acute pancreatitis (SAP) is a recognized cause of intra-abdominal hypertension (IAH), often resulting in secondary complications such as respiratory failure, renal dysfunction, and hemodynamic instability. Nevertheless, its role in increasing ICP and causing acute anisocoria has been minimally documented in the existing literature.

The present report examines the development of acute-onset anisocoria and increased ICP secondary to elevated IAP. In the ICU, the sudden onset of anisocoria is frequently indicative of intracranial pathology, often associated with increased ICP and subsequent compression of the third cranial nerve, necessitating urgent intervention to mitigate ICP.[1,2] However, it is imperative to recognize that anisocoria is not exclusively attributable to CNS causes; a comprehensive understanding of its diverse etiologies is crucial for optimal management. Furthermore, the presence of a normal computed tomography (CT) scan of the brain may suggest alternative underlying mechanisms. This report details 3 cases of acute anisocoria associated with increased ICP in patients suffering from SAP. Early implementation of cerebroprotective strategies aimed at ICP reduction has been observed to facilitate the resolution of anisocoria. Unfortunately, the 1st patient did not survive, while the other 2 made a full recovery. These cases highlight the occurrence of acute anisocoria as a result of elevated ICP due to increased IAP.

2. AIM

This case series aims to explore the association between elevated IAP and ICP, as clinically reflected by anisocoria. It also seeks to evaluate the effectiveness of early cerebroprotective interventions including osmotherapy, diuretic therapy, and IAP-lowering strategies in reducing signs of raised ICP, with particular focus on the resolution of anisocoria as a clinical indicator of therapeutic response.

3. CASE STUDY

3.1 Case study 1

A 22-year-old male student was admitted to the ICU during the 1st week of gallstone-induced SAP, complicated by respiratory failure and acute kidney injury. On initial examination, the patient was afebrile, restless, and exhibited tachypnea. Vital signs were as follows: blood pressure of 150/90 mm Hg, heart rate of 140 bpm, respiratory rate of 35 breaths per minute, and oxygen saturation of 88% on high-flow nasal cannula (HFNC). Abdominal examination revealed distension and diffuse tenderness. IAP, measured via the intravesical method, was 20 mm Hg.

The patient was subsequently intubated and commenced on mechanical ventilation. Laboratory investigations demonstrated leukocytosis, deranged liver function tests, renal impairment, and severe metabolic acidosis as revealed by arterial blood gas analysis. Fluid resuscitation was managed conservatively, targeting a central venous pressure (CVP) less than or equal to 12 mm Hg. Renal support was provided through alternate-day hemodialysis. Multiple CT guided percutaneous drainage procedures, including trans-gastric and pelvic approaches, were performed. A Ryle's tube was placed and maintained on continuous aspiration for decompression. On the 4th day of admission, clinical assessment revealed anisocoria, characterized by a dilated and non-reactive right pupil, while the left pupil remained normal in size and reactive to light (Figure 1). At that time, blood pressure was 160/100 mm Hg, and pulse rate was 90 bpm. Given the concern for raised ICP, optic nerve sheath diameter (ONSD) was measured and found to be 5.5 mm, sug-

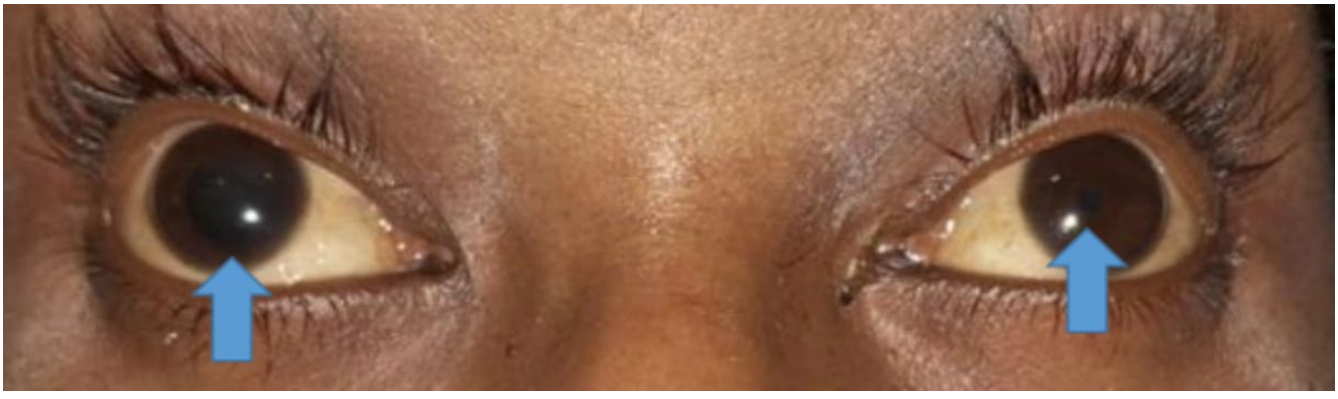

Figure 1. The right pupil is dilated compared to the left as shown by the blue arrow.

gestive of elevated ICP. Simultaneously, IAP had increased to 26 mm Hg. Consequently, osmotherapy was initiated to reduce ICP, and a flatus tube was inserted to relieve abdominal compartment pressure. An urgent cranial CT scan was performed, which showed no acute intracranial pathology.

A gastroenterology consultation was obtained to evaluate the potential need for exploratory laparotomy; however, surgical intervention was deferred as the patient was still within the early second week of SAP, a phase typically managed conservatively. A neurology consultation was also sought, and further recommendations for ICP management were implemented. Despite aggressive multidisciplinary interventions, there was no resolution of anisocoria. Unfortunately, the patient's condition continued to deteriorate, and he ultimately succumbed to the illness the following day. Notably, both anisocoria and elevated IAP persisted until the time of death.

3.2 Case study 2

A 55-year-old male diagnosed with biliary pancreatitis was admitted to the ICU during the 2nd week of illness due to progressive respiratory distress and abdominal distension. In light of worsening tachypnoea and hypoxemia, the patient was intubated and placed on mechanical ventilation. On admission, his vital signs were as follows: blood pressure of 180/90 mm Hg, pulse rate of 130 bpm, respiratory rate of 34 breaths per minute, and oxygen saturation (SpO_2) of 87% on room air. Measurement of IAP revealed an elevation to 21 mm Hg, consistent with IAH. Subsequently, the patient underwent CT-guided trans-gastric percutaneous drainage under sedation and analgesia. During transport back from the radiology unit, the patient developed sinus bradycardia, accompanied by a blood pressure of 170/100 mm Hg. As part of the routine post-sedation neurological evaluation, a pupillary examination was performed by the resident physician. The right pupil was noted to be dilated yet reactive to light, as illustrated in Figure 2. The left pupil remained normal in size and reactivity. No focal neurological deficits or localizing signs were identified at that time.

Laboratory investigations revealed normal acid-base status and serum electrolyte levels. In view of the patient's significant coagulopathy and persistently elevated blood pressure, an urgent non-contrast CT scan of the brain was performed to exclude intracranial hemorrhage. The imaging findings were unremarkable. Given the potential for increased ICP, we assessed the ONSD, which was recorded at 5.3 mm, indicating raised ICP. Furthermore, IAP remained elevated at 23 mm Hg. Aggressive cerebroprotective measures were promptly initiated, including head elevation, mannitol infusion, and administration of furosemide. A nasogastric tube was maintained for continuous drainage, while a flatus tube was inserted to alleviate IAP. Over time, the patient's hypertension and bradycardia gradually improved, and after 48 h, the right pupil returned to normal size. The patient demonstrated consistent progress, was successfully extubated, and was later transferred to the ward.

3.3 Case study 3

A 42-year-old male was transferred to our hospital from a private facility with a diagnosis of acute pancreatitis. He had no prior history of any chronic illness. Upon examination, he was agitated and restless, with a temperature of 38.9°C. Physical assessment revealed abdominal distension and tenderness on palpation. The remaining examinations were within normal limits. At the time of admission, his vital signs were as follows: blood pressure 90/60 mm Hg, pulse rate 128 bpm, respiratory rate 26 breaths per minute, and oxygen saturation at 94% on 5 L of oxygen via face mask. Arterial blood gas analysis indicated metabolic acidosis with type 1 respiratory failure.

Laboratory investigations revealed elevated pancreatic enzyme levels, leukocytosis, anemia, mildly deranged liver enzymes, and raised inflammatory markers. An abdominal ultrasound was suggestive of acute severe pancreatitis. The patient received treatment comprising intravenous antibiotics, IV fluids, analgesics, and respiratory support via BiPAP, along with various supportive and symptomatic interventions. Despite these measures, the patient continued to experience abdominal distension, with an IAP recorded at 20 mm Hg using the intravesical method. On the 13th day of illness, the patient developed severe headache and exhibited signs of increased ICP, such as hypertension, bradycardia, and drowsiness, although he remained able to follow commands.

Given our prior experience with 2 cases of elevated ICP, we promptly assessed his pupils. The left pupil was dilated but reactive to light, while the right pupil was normal

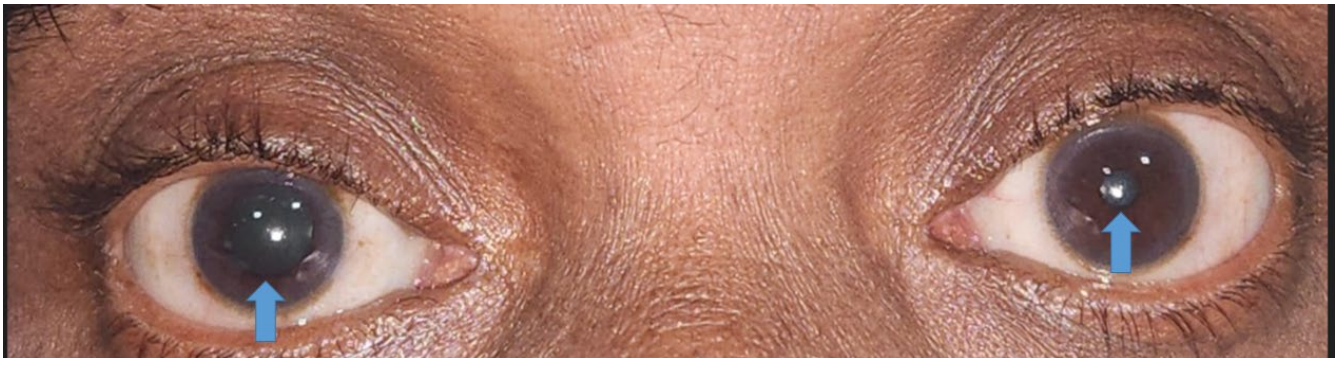

Figure 2. The right pupil is dilated in comparison to the left as shown by the blue arrow.

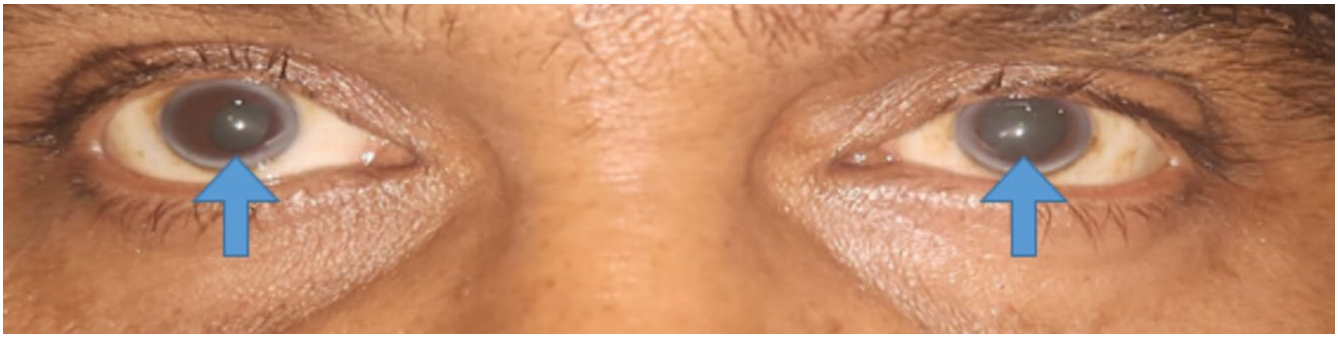

Figure 3. The left pupil is dilated in comparison to the right as shown by the blue arrow.

in size and reactive, as shown in Figure 3. Suspecting increased ICP, we evaluated the ONSD, which measured 5.2 mm suggestive of elevated ICP. Concurrently, the IAP was reassessed and found to be further elevated at 22 mm Hg. We swiftly implemented aggressive cerebroprotective measures, including head elevation, administration of mannitol and furosemide to reduce ICP, continued nasogastric drainage, and the introduction of a flatus tube to relieve IAP. A CT scan of the head was performed to exclude intracranial hemorrhage, but the imaging revealed no abnormalities. After 36 h, the patient demonstrated marked improvement in mental status, and the left pupil returned to normal size and reactivity. The patient made remarkable progress and was ultimately discharged from the hospital.

4. DISCUSSION

Acute anisocoria may arise from various underlying mechanisms, including damage to the parasympathetic postganglionic fibers, degeneration of the ciliary ganglion, or aberrant reinnervation of the pupilloconstrictor muscles, all of which contribute to disruptions in normal pupillary function.[3,4] While many cases are idiopathic, potential etiologies include infectious, inflammatory, ischemic, and perfusion-related disturbances within the orbital region, as well as the effects of general anesthesia.[3–6]

The occurrence of anisocoria in the setting of SAP is exceedingly rare, with only 1 prior case report documenting such an association. However, in that instance, the underlying etiology was believed to be unrelated to pancreatitis and instead attributed to conditions such as sarcoidosis, Guillain–Barré syndrome with cranial nerve involvement, or a possible viral infection.[5] In the present case, anisocoria may have resulted from perfusion abnormalities or an imbalance in ciliary ganglion function secondary to ischemia, likely precipitated by elevated blood pressure. Additionally, the potential role of systemic cholinergic nerve susceptibility, exacerbated by the administration of midazolam and fentanyl, cannot be overlooked.[7]

Elevated IAP triggers a series of physiological alterations that ultimately result in increased ICP. The increase in IAP leads to the upward movement of the diaphragm, which in turn raises intrathoracic pressure and central venous pressure. This rise in pressure causes venous compression, a decrease in wall compliance, and an expansion of the inferior vena cava (IVC). Consequently, intracerebral venous pooling occurs, which hampers venous outflow from the brain. The diminished venous outflow subsequently lowers cerebral perfusion pressure, contributing to a gradual increase in ICP. These interrelated mechanisms illustrate the influence of elevated IAP on cerebral hemodynamics and emphasize the necessity of monitoring and managing IAH to avert secondary neurological complications.[8,9] The noted rise in ICP may have contributed to the onset of acute anisocoria. In this instance, ICP was evaluated indirectly through the measurement of ONSD. The theory of increased ICP is further corroborated by the existence of related clinical symptoms, such as sinus bradycardia, hypertension, and an irregular respiratory pattern, all of which align with indicators of intracranial hypertension.

These cases highlight the need for further investigation into the pathophysiological mechanisms linking systemic conditions such as severe pancreatitis with acute anisocoria. A deeper understanding of these associations may help refine diagnostic approaches and therapeutic interventions in critically ill patients.

In our series, all patients presented with high blood pressure, and notably, hypertension was documented at the time anisocoria was first detected. A comprehensive and systematic evaluation was undertaken to exclude other potential etiologies of anisocoria. Intracranial pathologies such as oculomotor nerve palsy, compressive lesions (including aneurysms), traumatic brain injury, and brain herniation were carefully ruled out. Potential alternative causes were also excluded, including Adie's pupil, pharmacologic or toxic effects, prior ocular trauma or surgery, and the use of mydriatic eye drops. These cases highlight the critical importance of conducting a thorough pupillary examination in patients with SAP, particularly when clinical indicators of elevated ICP, such as anisocoria, are present, even in the absence of abnormalities on neuroimaging like CT. Timely and urgent intervention to reduce ICP through conservative measures including osmotherapy, diuretic administration, and strategies aimed at lowering intra-abdominal pressure may mitigate the deleterious effects of raised ICP. This approach contributed to favorable clinical outcomes, as demonstrated in cases 2 and 3. However, accurately identifying the threshold of IAP that triggers a rise in ICP remains challenging without direct ICP monitoring.

6. CONCLUSIONS

(1) In patients with SAP, a rise in IAP can elevate ICP, potentially manifesting as anisocoria.
(2) ONSD measurement is a valuable, non-invasive tool for detecting elevated ICP, even when CT findings of the head are normal.
(3) Timely cerebroprotective measures can improve patient outcomes.

Conflict of interest

We declare that we do not have any conflict of interest.

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None.

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