Case Report

# Postoperative acute anisocoria and old traumatic brain injury

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## ABSTRACT

Anisocoria is an uncommon entity in general postoperative intensive care. We present a case of a 45-year-old man suffering from severe acute pancreatitis with a past history of traumatic brain injury (TBI), who developed hypertension, bradycardia and anisocoria soon after re-exploration surgery under general anaesthesia. Computed tomography showed no new lesion. Measures directed towards reducing intracranial pressure resulted in amelioration in about 12 h. The possible role of old TBI in the causation of anisocoria during general anaesthesia and resuscitation has been explored in this report.

**Key words:** Anisocoria, postoperative period, traumatic brain injury

## INTRODUCTION

Causes of anisocoria include compression or destruction of the third cranial nerve by increased intracranial pressure (ICP) from tumour, thrombus, oedema, aneurysm or haemorrhage.[1] Cases of anisocoria have rarely been reported in the postoperative intensive care unit (ICU) setting. We report a case of anisocoria in ICU that was in all likelihood linked to the vulnerability of brain with old traumatic brain injury (TBI) to regional increase in ICP secondary to general anaesthesia and intraoperative fluid resuscitation.

## CASE REPORT

A 45-year-old male with severe acute pancreatitis (SAP) was referred to the ICU with acute physiology and chronic health evaluation (APACHE II) score 20 and intra-abdominal hypertension. About 5 years back, he had had a TBI (Marshal grade III)[2] with significant intracranial bleed. It was a focal lesion and he had a complete clinical recovery with conservative management. After surgical necrosectomy performed in the sixth week of SAP, there was fresh bleeding from the drains requiring an urgent re-exploration. His haemoglobin dropped from 10 gm% to 6 gm% within few hours, and his prothrombin time was 6 s prolonged, with a platelet count of 60,000/cmm. General anaesthetics included isoflurane, besides fentanyl, thiopental, midazolam and vecuronium bromide. The intraoperative period was uneventful from the point of adverse events like hypoxia, hyperventilation, severe hypotension (mean blood pressure <60) or hypertension. However, he required significant volume resuscitation (about 5 L in 2 h) comprising of 1.5 L of crystalloid solution, 0.5 L of colloid solution, 4 units of packed red blood cells (PRBC), 4 units of fresh frozen plasma (FFP), 10 units of random donor platelet and 10 units of cryoprecipitates, to maintain haemostasis and haemodynamics. Soon after shifting him back to the ICU, he developed sinus bradycardia with hypertension (150/90 mmHg). Pupillary examination revealed left-sided pupillary dilatation with minimal reaction to light. The right pupil was normal. Difference in pupillary size was more than 3 mm. Fundoscopy did not reveal papilloedema. His acid base and electrolytes were normal. A negative pilocarpine test ruled out Adie syndrome and he did not exhibit features of Horner's syndrome. Absence of any appreciable localizing sign was suggestive of a nonvascular major intracranial event as the cause

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of acute anisocoria. In the background of existing coagulopathy, intracranial haemorrhage was kept as a probable diagnosis. But, an urgent computed tomography scan revealed an old, healed haemorrhage in the left fronto-temporal region as sequelae to the past TBI and no new organic lesion or bleed [Figure 1]. Soon thereafter, interventions were started to decrease ICP and 1 g/kg intravenous bolus mannitol and 20 mg frusemide were administered. Repeat doses were given every 4-hourly for 24 h and he was nursed in the head-up position. Within 12 h of initiating measures for ICP reduction, hypertension and bradycardia resolved and his left pupil started to decrease in size and reaction to light was restored. After normalization of pupillary size and response to light, mannitol and frusemide were stopped. On day 3 of the event, he was fully conscious and his trachea was extubated.

## DISCUSSION

Anisocoria during general anaesthesia has been reported mostly during induction of anaesthesia on account of parasympathetic dominance by anaesthetic drugs such as thiopental, midazolam, isoflurane and vecuronium induced autonomic tone disturbance.[3-7] Lazar *et al*.[5] reported that midazolam can cause a brief ischemic event in the left cerebral hemisphere in patients with an associated abnormality of the left side of the brain. Although thiopental, midazolam, isoflurane and vecuronium were administered in our patient, anisocoria appeared during recovery from general anaesthesia and not during the induction period. The triad of acute onset hypertension, sinus bradycardia and anisocoria is suggestive of regional increase of ICP. Easy reversibility of the triad after a short period of mannitol and frusemide therapy also justifies the transient rise in regional ICP as cause of anisocoria in our case. Haemodynamically, volume resuscitation of 5 L in a short period of 2 h was well tolerated by the patient and the central venous pressure (CVP) never went beyond 10 mmHg.

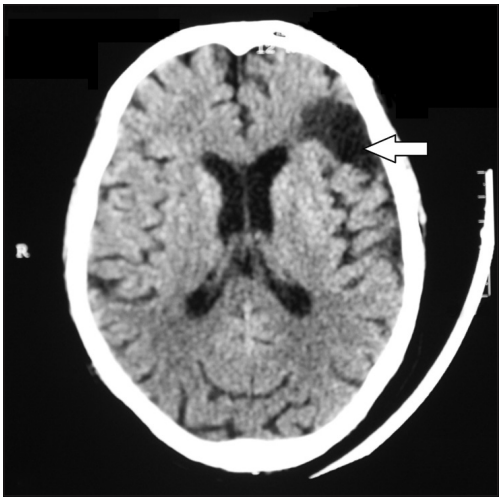


**Figure 1:** Computer tomography scan of the brain with old left frontotemporal infarct (arrowed)

Following TBI, an increased CVP occurring with aggressive crystalloid resuscitation may contribute to the loss of brain compliance and the development of intracranial hypertension.[8]

In our case, this was probably not the underlying mechanism. Rather, TBI has been reported to give rise to latent occult "cryptic" vascular lesions.[9] We postulate that an old TBI may render a brain vulnerable to a regional increase in ICP during active fluid resuscitation, which may also get influenced by the autonomic tone disturbance secondary to drugs like thiopental, isoflurane and vecuronium by the following mechanisms:

1. Clinically latent occult "cryptic" vascular lesions may persist for an unidentified period after TBI.
2. These "cryptic" vascular lesions may have much autoregulatory dysfunction.
3. Such lesions may cause regional elevation of ICP during fluid resuscitation with or without general anaesthetics.

Although anisocoria has been considered as a benign event in a critical care patient,[10] our case suggests that anisocoria in a subset of the population may actually be an impending neurologic emergency. Authors think it appropriate to believe that one should exert extra caution about the rapidity of fluid resuscitation in the management of a case of old TBI. Also, it may be advised that fluid resuscitation in such patients is accompanied by concomitant pupillary examination.

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