Vol 4 | Issue 19 | 2023

Interesting cases NEUROLOGY

Neurology Case Series

Vol 4 | Issue 20 | 2023

Dear Doctor,

Greetings from Micro Labs Limited.

We are happy to bring you "Neurology case series", which contains latest and interesting cases in the field of Neurology.

This issue contains

1. **A rare case report on transorbital penetration injury.**
2. **An unusual case of hypernatremia-related anorexia with osmotic demyelination syndrome.**
3. **Case report on anisocoria without extraocular muscle dysfunction due to mild traumatic brain injury with a midbrain contusion.**
4. **Case report on Invasive and metastatic version of meningioma**

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team,

MICRO LABS SYNAPSE
Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

1. A rare case report on transorbital penetration injury

Introduction:

Transorbital penetration injury (TOPI) can cause significant neurological symptoms due to damage to important neurovascular structures around penetration routes.[1] Penetrating brain injuries were lethal because of damage to critical systems such as the vascular system and the brainstem, as well as the possibility of infection. The risk of infection was probably higher in the case of TOPI induced by wood material.[2] Because every person's orbital architecture and cerebral vascular anatomy were different, accurately identifying the perforation pathway was essential to prevent serious complications. In this case, a comprehension of the anatomy around the invading object in three dimensions (3D) was necessary. The inferior rectus muscle had been injured in this instance; this conclusion was supported by the 3D printer model (Figure 1).[1] This case report described a transorbital penetration injury in which important brain regions were not damaged throughout the penetration pathway.

This case showed the significance of recognizing the neuroanatomical structures around the penetrating object. A 3D assessment of the trauma etiology was critical for determining the best treatment plan.

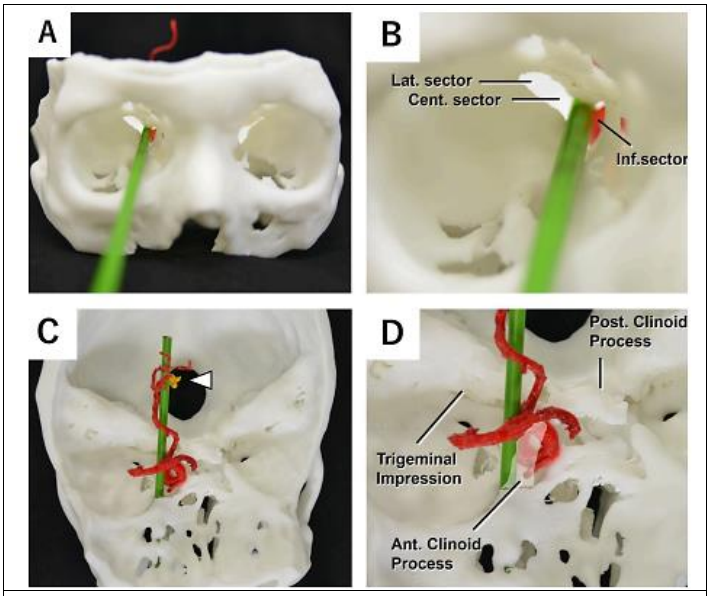

Figure 1: Imaging of a 3-D printer model on day 18. The lower sector of the right superior orbital fissure was traversed by the gardening pole (A, B). Right posterior cerebral artery aneurysm development at the point of contact with a penetrating object (white arrowhead) (C). It was anticipated that the gardening pole would have entered the cavernous sinus on its right lateral side (D).

Case Presentation:

A 52-year-old man was injured in his right eye after a gardening pole percutaneously pierced the medial side of his right infraorbital area. He had collapsed in his garden after consuming too much alcohol. His wife removed the gardening pole from his head as soon as she saw the injuries. After that, he was admitted to hospital's emergency department. Upon physical examination, a 4-mm scar on the right eye's lower lid was noticed (Figure 2).

He had altered consciousness since his Glasgow Coma Scale score was 8. The right ambient cistern's subarachnoid haemorrhage was shown by the head computed tomography (CT) scan. Similar

Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

subarachnoid haemorrhage and brain contusion were seen in the medial posterior region of the right temporal lobe on magnetic resonance imaging (MRI). There were no vascular abnormalities identified by cerebral angiography or CT (Figure 3A). Consequently, cautious blood pressure treatment was started along with conservative management. The patient became conscious on the second day, allowing for more neurological testing. There were no complaints related to the visual field or eyesight, however a little limitation in the eyeball's downward movement was noted during the physical assessment of eye movement. Two-dimensional data of the skull and intracranial arteries were extracted using Ziostation.

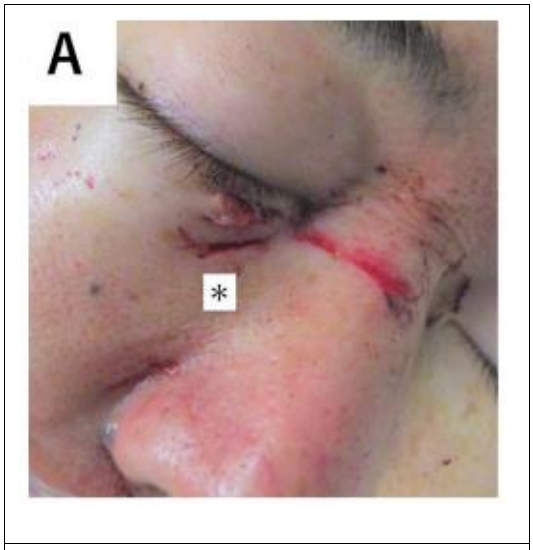

Figure 2: The skin of the skull was pierced by the gardening poles. The lower lid of his right eye was the entry point (*).

This data was then rebuilt into a 3D file and transferred to the stereolithography (STL) format. The 3D model demonstrated how the penetrating object entered through the right superior orbital fissure's inferior sector, travelled through the lateral space to the internal carotid artery, made contact with the ambient cistern's right posterior cerebral artery (PCA), and ultimately arrived at the medial aspect of the occipital lobe. After 17 days of close observation, the patient's MRI revealed an aneurysm in the right PCA near the point of contact with the penetrating object. A 6.4-mm aneurysm in the right PCA P3 segment was identified through cerebral angiography (Figure 3B). Since selective embolization of the aneurysm was thought to be difficult, endovascular parent artery occlusion was carried out after an occipital artery (OA)-PCA bypass to protect the blood supply (Figure 3C).

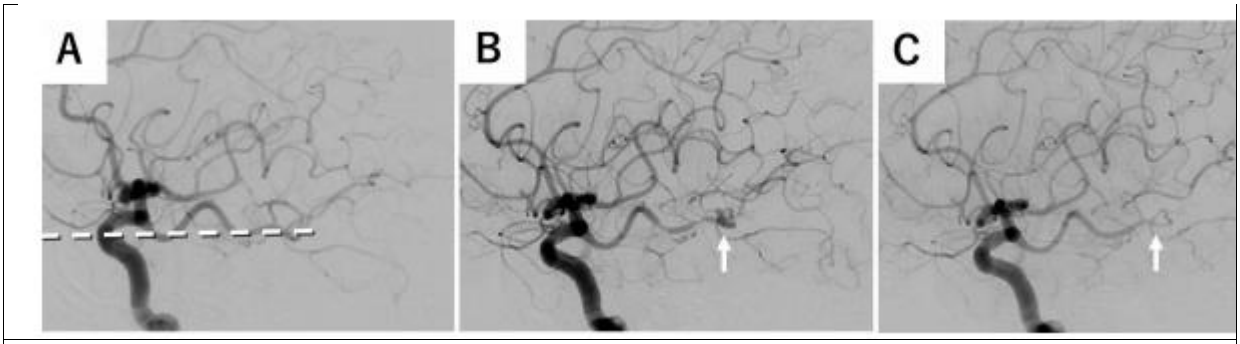

Figure 3: The results of cerebral angiography for vascular abnormalities. The white dotted line represents the gardening pole's simulated entrance path (A). On day 17, cerebral angiography reveals an aneurysm (white arrow) at the beginning of the calcarine artery (B). The aneurysm (white arrow) has vanished following endovascular parent artery occlusion (PAO) on day 33 (C).

An MRI after surgery revealed no evidence of a brain infarction. Meningitis or aneurysm rupture were not present when the patient was discharged from the hospital. The patient

experienced left superior homonymous quadrantanopia after surgery, which resolved spontaneously within 6 months.

Discussion:

In the current case, the patient's skull was pierced by a gardening pole in an unexpected direction, but with the right care, the neurological abnormalities were reduced.[1] J.A. Morales-Gomez et al. showed that anatomically, the superior orbital fissure was divided into inferior, middle, and lateral sectors. The inferior rectus muscle, abducens nerve, and nasociliary nerves were located in the inferior sector of the superior orbital fissure.[3] In this case, it was noted that the inferior rectus muscle had been injured; this conclusion was supported by the 3D printer model.[1]

Conclusion:

This study experienced an uncommon penetrating head injury case with little consequences. This case demonstrated how crucial it was to understand the neuroanatomical structures around the target of penetration. A 3D study of the trauma etiology was critical for determining the best treatment plan.

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MICRO LABS SYNAPSE
Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

2. An unusual case of hypernatremia-related anorexia with osmotic demyelination syndrome

Introduction:

Osmotic demyelination syndrome (ODS) is a non-inflammatory demyelinating disorder. ODS symptoms range from tremors and hemiparesis to encephalopathy and death.[1] ODS was characterized by myelin sheath injury in brain cells caused by sudden osmotic changes in the plasma. Magnetic resonance Imaging (MRI) based studies reported a 0.3-1.1% rate of hospital admissions. The most common cause of ODS was rapid correction of hyponatremia.[2] Other osmotic abnormalities, such as hypernatremia can cause this condition, particularly in individuals with pre-existing comorbidities such as malnutrition. Wernicke-Korsakoff syndrome (WKS) was another neurological disorder caused by thiamine deficiency that was most common in alcoholics. Aside from drinking, disorders related with poor nutrition, such as malnutrition or schizophrenia, may also contribute to WKS. Anorexia was defined by extremely low body weight resulting from inadequate energy intake. Anorexia occurs in various mental disorders, such as major depressive disorder.[1] This was a case of anorexia caused by a severe depressive disease, with signs and symptoms of hypernatremic ODS and WKS in the context of chronic malnutrition.

Physicians should evaluate the co-occurrence of ODS and WKS in malnourished patients. Thus, electrolyte imbalances and dietary deficiencies in malnourished individuals should be rectified as soon as possible to avoid irreversible neural consequences.

Case Presentation:

A 39-year-old female admitted at the emergency room because of the complaints of generalised weakness and decreased awareness. She suffered from anorexia for seven months after the deaths of her mother and sister, which exacerbated in the last two months. According to the patient's friend, she lost 30 kg in the previous seven months and started experiencing paranoid thoughts and auditory hallucinations. Her friend also said that she experienced depressed mood, lethargy, and anhedonia in the previous seven months, all of contributing factors to severe depressive illness. She suffered from psychotic symptoms (such as thoughts of poisoning and auditory hallucinations), which raise the probability that she had a premorbid mental illness (such as schizophrenia) that became worse after lost her loved ones. The patient could have been a suitable candidate for electroconvulsive treatment because of

Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

her likely severe depression, but her unstable state made it impossible. Her oxygen saturation was 90% in room air and 96% with an oxygen mask when she was admitted. Her temperature was 36 degrees Celsius, her blood pressure was 90/60 mmHg, and her pulse rate was 60 beats per minute. On neurologic examination, the patient was awake, attentive to her name, and followed orders, but she was not responsive to questions. Her upper and lower limbs have a symmetrical force of 2/5. She also had less deep tendon reflexes in her lower limbs. The laboratory results upon presentation were 15,600 WBC/µL for white blood cells, 7.8 g/dl for hemoglobin, 96 fl for mean corpuscular volume, and 540 $\times 10^3$ /µL for platelets. In addition, her investigation revealed the following: alkaline phosphatase (261 U/L), LDH (1568 U/L), BUN (68 mg/dl), creatinine (0.7 mg/dl), lactate (20 mg/dl), calcium (8.1 mEq/L), phosphorus (2.2 mEq/L), magnesium (1.5 mEq/L), and hypernatremia (179 mEq/L) and hypokalemia (2.1 mEq/L). Her computed tomography scan showed no evidence of ischemic or hemorrhagic stroke. Eryphalitis and meningitis were ruled out based on the results of the lumbar puncture. MRI was performed since the diagnosis was unclear. The most likely diagnosis, based on the radiologic findings, laboratory findings, and history, were WKS and ODS. MR scans with multiplanar images in various pulse sequences indicated symmetric aberrant signal intensities in the medial aspect of both thalami, bilateral external capsule, pons, and cerebellar hemispheres, as well as limitation in DW images, indicating ODS (Figure 1).

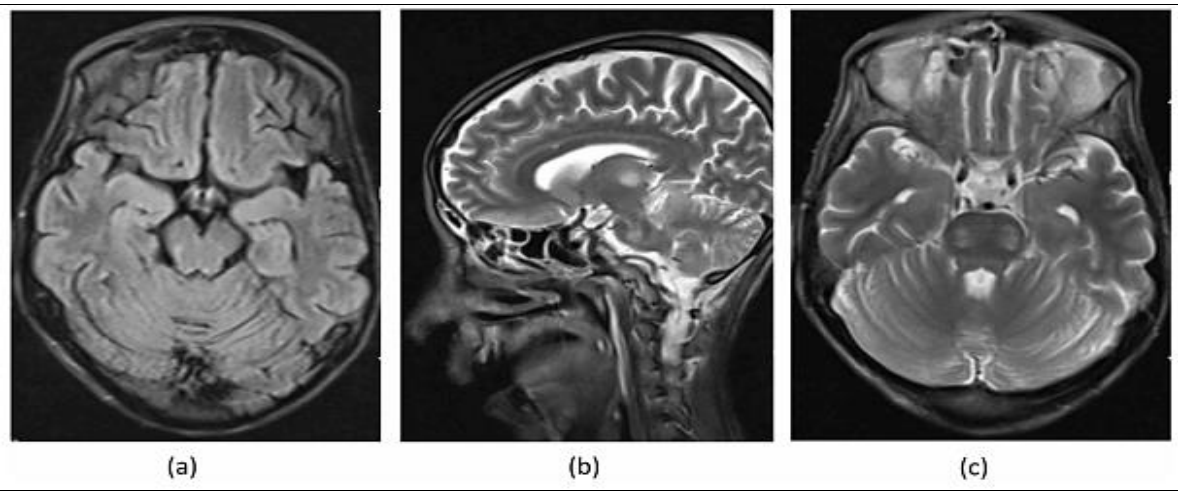

Figure 1: Symmetric aberrant signal intensities in DW images that were indicative of ODS in the medial portion of both thalami, bilateral external capsule, pons, and cerebellar hemispheres with limitation (a, b). Bilateral medial thalami involved symmetrically, suggesting WKS, and extrapontine involvement, suggesting ODS (c).

No hydrocephalus was visible on the MRI, and every ventricle was symmetric and functioning normally. The paranasal sinuses were unremarkable, and the optic chiasm, pituitary stalk, gland, and visible sections of both orbital contents had normal signals and appearances. Serum treatment (normal saline 1000 ml twice daily) was initiated for the patient. Serum lactate levels that were elevated and decreased awareness both indicated to systemic infections, or severe sepsis. Therefore, the patient was started antibiotics (vancomycin: 1 g loading dosage, followed by 1 g BID, and meropenem: 1 g loading dose, followed by 1 g TDS). Over the course of six days, sodium levels steadily decreased to 149 mEq/L (Figure 2).

Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

Over the course of three days in the hospital, potassium levels increased to 3.7 mEq/L. A nutritionist's advice was obtained before administering additional micronutrients including zinc, copper, and selenium. Because of the MRI evidence of WKS, a therapeutic dose of thiamine (1 g IV BID) was also started on day 1. Even with appropriate dietary and electrolyte treatment, the outcomes were inadequate. Additionally, the patient did not respond to antibiotics. A blood culture test performed on day 4 revealed multidrug-resistant Klebsiella pneumonia. The patient started taking colistin, which was administered as a loading dosage of 9,00,000 IU followed by 4500,000 IU BID. On the day 10 of hospitalization, the patient's condition worsened due to the severe sepsis condition, which also caused multiorgan failures such cardiac arrest and a lowered mental state. Despite successful cardiac resuscitation, the patient finally passed away due to brain death.

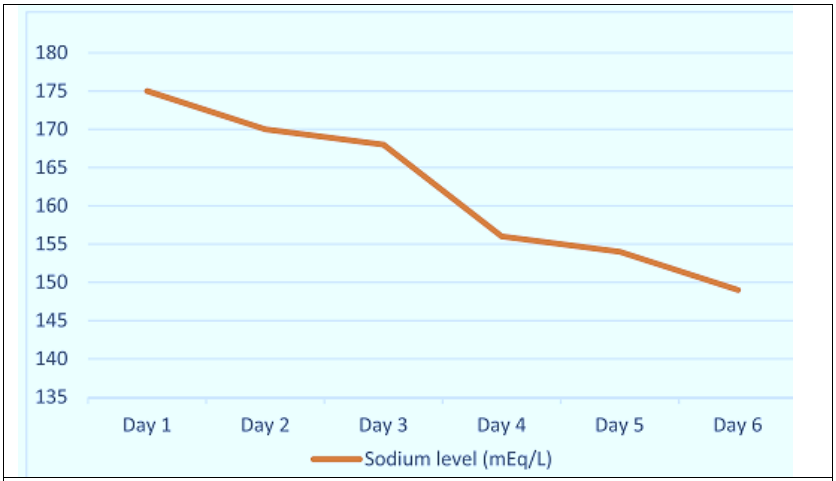

Figure 2: Serum sodium levels with time.

Discussion:

To date, the most common condition associated with ODS has been rapid correction of hyponatremia.[1] In a previous study by Ghosh et al. found that other osmotic abnormalities, such as hypernatremia and hypokalaemia, may result in ODS.[3] Furthermore, Lambeck et al. demonstrated that patients with comorbidities such as chronic alcoholism and malnutrition were more likely to develop ODS due to underlying electrolyte abnormalities.[4]

Conclusion:

Hypernatremia has been proposed as a rare cause of ODS. When additional comorbidities (e.g., malnutrition) are present, the risk of hypernatremic ODS increases. Thiamine deficiency may result in WKS that overlaps with ODS in malnourished individuals. To avoid catastrophic medical and behavioral problems, primary care physicians need to recognize early indicators of severe depressive illness. In addition, careful monitoring of electrolytes and vitamins, as well as early diagnosis of neurological signs, were essential in managing anorexia patients.

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Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

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Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

3. Case report on anisocoria without extraocular muscle dysfunction due to mild traumatic brain injury with a midbrain contusion

Introduction:

Anisocoria is a significant clinical sign of potentially fatal cerebral damage that rarely manifests as isolated mydriasis

Anisocoria and mydriasis are critical neurological findings that can indicate possibly life-threatening diseases. Perioperative pupil size evaluation was critical for anesthesiologists to understand the patient's current disease, medical history, and medication usage; this examination was especially important for patients scheduled for neurosurgery.[1] Anisocoria and a lower baseline Glasgow Coma Scale (GCS) score have been linked to significant impairment and poor recovery from traumatic brain injury (TBI). Anisocoria was often caused by either impaired pupil constriction (parasympathetic route) or impaired pupil dilation (sympathetic pathway). With the population's age, emergency physicians were going to treat more geriatric TBI patients, with or without anisocoria.[2] This case report described the condition of an old Japanese woman who had a mild traumatic brain injury (TBI) and midbrain hemorrhage, but did not have anisocoria or paralysis of the extraocular muscles.

Case Presentation:

A 79-year-old woman was admitted to the hospital soon after falling from bicycle. The patient had a history of gastroesophageal reflux disease and hypoacusis, which necessitated the use of hearing aids, and had been prescribed esomeprazole 10 mg once day and mosapride citrate hydrate 5 mg three times daily. When the patient arrived at the emergency room, her vital signs were as follows: blood pressure of 150/79 mmHg; temperature of 35.8°C; heart rate of 58 beats per minute with regular rhythm; breathing rate of 20 breaths per minute; and oxygen saturation of 100% with supplementary oxygen given at 10 L/min via a basic oxygen mask. When the patient arrived, her Glasgow Coma Scale score was 11 (E3V3M5), which indicated impaired consciousness as a result of brain damage. Upon examination, the patient showed signs of fatigue but reacted to pain. Auscultation revealed that the trachea was central and that there were no reduced breath sounds or crackles. There was no abdominal distention. Drowsiness made cranial nerve examination difficult. There was a bruise near the right eye. The pupil sizes varied, measuring 3.0 mm on the left and 4.5 mm on the right. Both eyes responded to light. Using an NPi-200 pupillometer, automated infrared pupillometry (AIP) and neurological pupil index (NPi) were used to test the pupillary light reflex (PLR). It demonstrated anisocoria with a dilated right pupil. A hemorrhagic contusion in the left temporal lobe and left pretectal region of the midbrain, formed by the cerebellar tentorium, was found on a computed tomography (CT) scan of the head (Figure 1).

Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

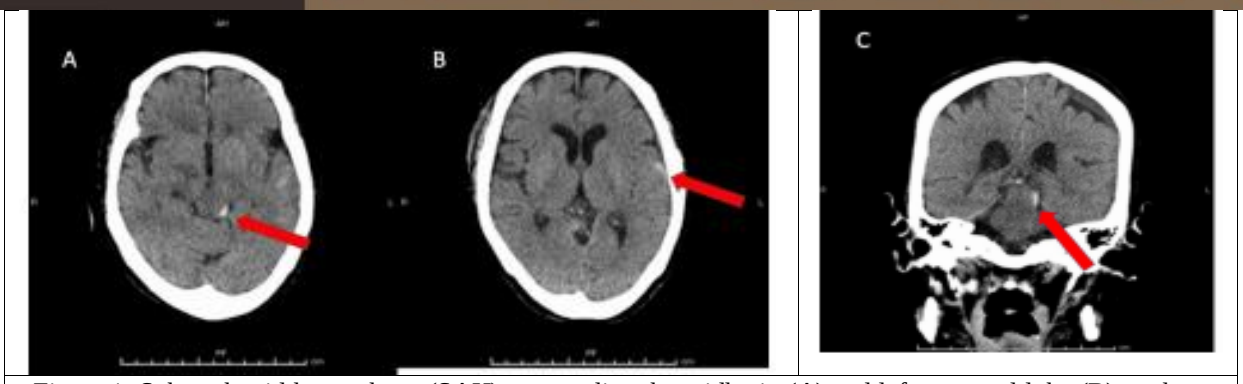

Figure 1: Subarachnoid hemorrhage (SAH) surrounding the midbrain (A) and left temporal lobe (B) as shown on axial computed tomography (CT). SAH and brain contusion are seen on a Coronal CT of the midbrain (C). The lesions are identified by red arrows.

There was also a pulmonary contusion, right clavicular fracture, and third and fourth rib fractures. A CT scan performed on the next day and on the fifth day in the hospital revealed no hemorrhage enlargement (Figure 2). The next day, cranial examinations revealed normal findings aside from anisocoria (right pupil, 4.5 mm; left pupil, 3.0 mm; reactive to light).

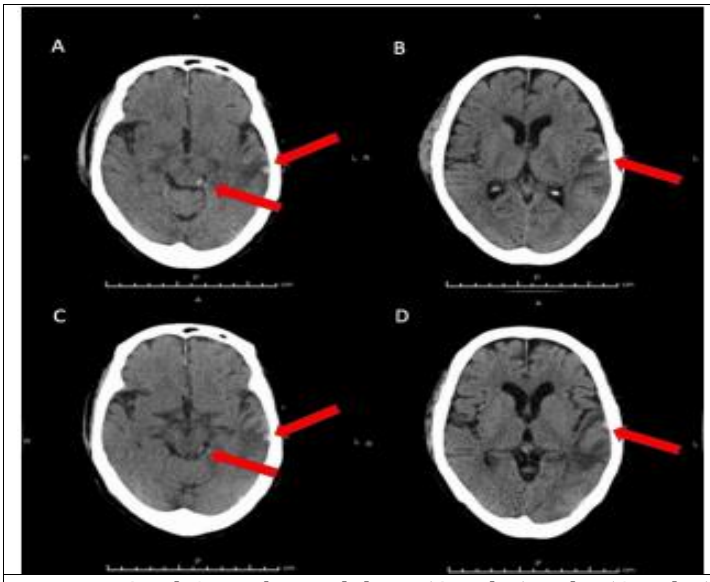

Figure 2: Axial CT on hospital day 2 (A and B) and 7 (C and D). There is no evidence of hemorrhage enlargement. The lesions are identified by red arrows.

Motility of the eyes was normal. Using an infrared pupillometer (NPi200; NeurOptics, Inc.), the NPi was 3.2 on the right and 4.4 on the left, with pupil diameters of 5.66 mm and 3.57 mm, respectively. The patient continued to have abnormal pupillary findings, with a difference in NPi of 2.0 on hospital day 5 and 1.5 on hospital day 7. On day 7 of hospitalization, the right clavicle fracture was treated with open reduction and plate fixation. Magnetic resonance imaging (MRI) of the head was done on hospital day 13 following an uneventful surgical course. The left midbrain and left temporal lobe contusions were confirmed by MRI. On day 17 of hospitalization, the patient was discharged from the hospital and was able to walk alone. At 4 and 32 days following discharge, follow-up visits revealed that the anisocoria persisted. She also reported experiencing dizziness and weight loss, but she was able to return to her normal routine without experiencing any visual disturbances. As of the most recent follow-up appointment, the NPi of the right eye had recovered to 3.8 and pupillary size had restored to symmetry between both pupils.

Discussion:

This case demonstrated anisocoria without significant extraocular muscle dysfunction as a result of mild TBI with contusion in the left pretectal region of the midbrain. In the present case, abnormal pupillary findings remained with an NPi difference of 2.0 on hospital day 5 vs 1.5 on hospital day 7.[2] In a previous study by Privitera CM et al. found that the presence of a difference in the NPi between pupils was another predictor of poor neurological outcomes.[3] Fortunately, with conservative therapy, the patient in this case recovered complete motor and mental functioning.[2]

Conclusion:

Anisocoria was a significant clinical sign of potentially fatal cerebral damage that rarely manifested as isolated mydriasis. We saw a patient who had anisocoria alone and no significant extraocular muscle dysfunction. This unusual presentation might be linked to midbrain damage.

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MICRO LABS SYNAPSE
Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

4. Case report on Invasive and metastatic version of meningioma

Introduction:

Meningioma is a frequent benign brain tumor that rarely invades neighboring tissues. Moreover, the tumor's invasive growth may increase the chance of a recurrence.

Meningiomas are the most prevalent non-malignant central nervous system (CNS) tumors, accounting for 38.3% of all cancers and 54.5% of non-malignant tumors in adults. In contrast, they are rare in children, with a reported incidence of 0.4%-4.6% of all primary CNS tumors in this age range.[1] This tumor generally occurs around the meninges; but, in rare cases, its invasion to adjacent tissues and organs such as various meningeal layers, brain vascular bed, and even skull has been documented. The chance of tumor recurrence may be increased as a result of the tumor's invasive expansion. Meningioma that was invasive and metastatic was rarely documented. Recurrent secondary meningioma that has spread to the pre-orbital soft and hard tissues was extremely rare.[2] This case demonstrated an unusual invasive meningioma that invaded deep orbital soft tissue after the initial tumor recurred.

Case Presentation:

The patient was a 56-year-old woman with a known case of meningioma who had radiation therapy and chemotherapy around 2 years ago, resulted in clinical improvement, but she returned with complaints of headache and acute exophthalmia. The patient's medical history showed that she was hypertensive with controlled state at the time of referral (systolic/diastolic blood pressures of 140/90 mmHg) and had no additional comorbidities. CT examination revealed a massive extra-axial, actively enhancing mass in the right side's anterior and middle cranial fossa, with invasion of the orbit, paranasal sinuses, and masticator, infratemporal, and buccal regions, resulting in the loss of neighbouring bony structures. The mass covered the right globe, resulting in significant proptosis. The mass exhibited restricted diffusion.

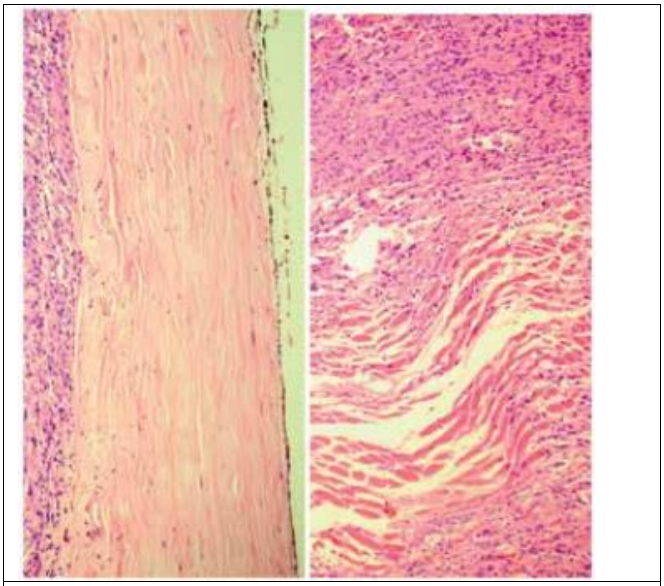

Figure 1: Histologic examination revealed that an invasive atypical meningioma invaded the optic nerve (left) and muscle bundles (right), H&E x 200

Vol 4 | Issue 19 | 2023
Interesting cases NEUROLOGY

The tumour surrounded the cavernous and supraclinoid portions of the right internal carotid artery. Craniotomy was a viable option for the patient. A white-gray bleeding mass measuring 7.5×6.0×4.0 cm^3 and affecting orbital soft tissue was discovered on macroscopical examination. The tumor covered the whole globe of the eye without involving any intraocular components, but it also reached the muscle bundles and deep orbital tissue. For pathological evaluation, a sample of the mass was submitted to a lab. Fragments of hypercellular neoplastic tissue were found with eosinophilic cytoplasm, occasionally occurring macro-nuclear nucleoli with mitotic figures approximately 4 to 5 numbers in 10 HPF, but no signs of necrosis suggesting atypical meningioma, the consequence of the primary malignant tumor recurrence (Figure 1). P53, Ki67, and epithelial membrane antigen (EMA) were all positive in the tumor sample when examined by immunohistochemistry (Figure 3).

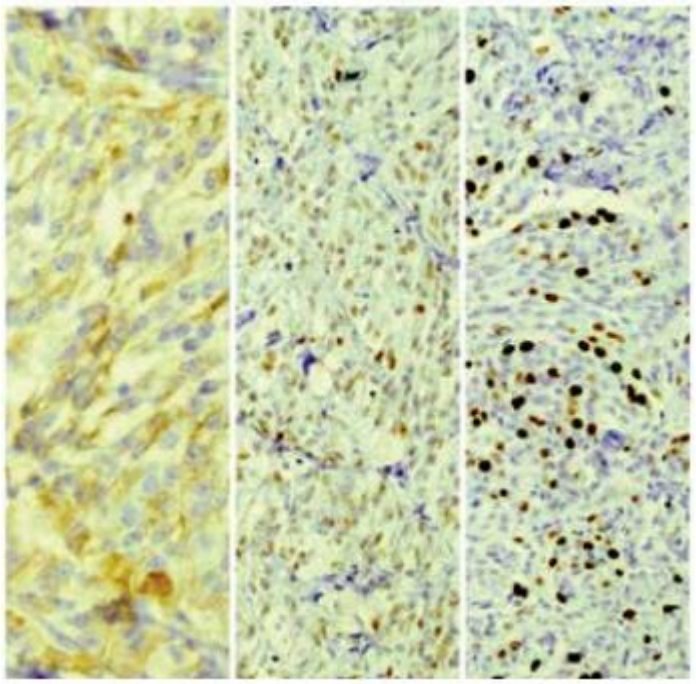

Figure 3. Positive epithelial membrane antigen (left), P53 staining (middle), and about 30% Ki-67 staining (right) were observed using immunohistochemical staining.

Discussion:

In this case, a recurring atypical meningioma with considerable extension to neighboring orbital tissues was observed, leading to a final diagnosis of invasive meningioma, grade II according to the WHO classification. Many previous cases demonstrating distinct invasive tendencies of these tumors have been observed.[2] In a previous study, Mrinal Matish et al. described an invasive meningioma with cystic intracranial tumor that invaded the left temporal fossa and extended into the middle and external ear.[3]

Conclusion:

Meningioma is a common benign brain tumor. It rarely invades nearby tissues but might stay asymptomatic. Moreover, the tumor's invasive development may increase the chance of a recurrence. Therefore, physicians should take into account the potential of the tumor and treat it quickly to avoid morbidity from tumor extension.

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