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Chapter

Lesions at the Foramen of Monro Causing Obstructive Hydrocephalus

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Abstract

The foramen of Monro has also been referred to by the name of interventricular foramen. The structures comprising this foramen are the anterior part of the thalamus, the fornix and the choroid plexus. Vital structures surround the foramen, the damage to which can be catastrophic leading to disability either temporary or permanent. In the literature it has been shown that tumors occurring in the area of interventricular foramen are rare and usually cause hydrocephalus. The operative approach depends upon the location of the tumor which can be either in the lateral or the third ventricle. Various pathologies which can lead to foramen of Monro obstruction and obstructive hydrocephalus include colloid cyst, craniopharyngioma, subependymal giant cell astrocytoma [SEGA], Neurocysticercosis, tuberculous meningitis, pituitary macroadenoma, neurocytoma, ventriculitis, multiseptate hydrocephalus, intraventricular hemorrhage, functionally isolated ventricles, choroid plexus tumors, subependymomas and idiopathic foramen of monro stenosis. In this chapter, we will discuss the various lesions at the level of foramen of Monro causing obstructive hydrocephalus and the management and associated complications of these lesions based on their type, clinical picture and their appearance on imaging.

Keywords: Foramen of Monro, interventricular foramen, obstruction, obstructive hydrocephalus, raised intracranial pressure

1. Introduction

The foramen of Monro has also been referred to by the name of interventricular foramen. The first description of this foramen was given by Alexander Monro in the year 1783 and 1797. The authors of that era were of the opinion that the use of nomenclature 'foramen of monro' was incorrect; instead 'interventricular foramen' would be more apt. Their reason was that Monro had interpreted the connection between lateral and the third ventricle in an incorrect way.

The structures comprising this foramen are the anterior part of the thalamus, the fornix and the choroid plexus. Vital structures surround the foramen, the damage to which can be catastrophic leading to disability either temporary or permanent.

The dimension of this foramen is not even 1 centimeter and thus the area present for any operative intervention is very small. It is not an easy task for the surgeons to excise the lesion as well as safeguard the vital surrounding structures at the same time.

In the literature it has been shown that tumors occurring in the area of interventricular foramen are rare and usually cause hydrocephalus. The operative approach depends upon the location of the tumor which can be either in the lateral or the third ventricle [1].

In this chapter, we will discuss the various lesions at the level of foramen of Monro causing obstructive hydrocephalus and the management and associated complications of these lesions based on their type, clinical picture and their appearance on imaging.

2. History and anatomy

2.1 Introduction

Fluid balance in central nervous system is basically maintained by cerebrospinal fluid [CSF], which is derived from blood and secreted by choroid plexus lining ventricles of the brain. CSF plays a major buoyancy role in the mechanical support to central nervous system.

Circulation of CSF through ventricular system of cerebral cortex takes place in such a way that there is free communication between cerebral and spinal subarachnoid compartment. It start from lateral ventricular cavities passing through foramen of Monro, then entering into third ventricle passing down along aqueduct of Sylvius and reaching fourth ventricle. The exit from fourth ventricle takes place through foramen of Luschka and foramen of Magendie to subarachnoid space around brainstem and spinal cord [2].

2.2 History

Foramen of Monro is named after a Scottish physician Alexander Monro Secundus [1733–1817], he was third son of Alexander Monro Primus and Isabella MacDonald. He matriculated at Edinburg University in 1745 and received his medical degree in 1755 [3]. He assisted his father Alexander primus in teaching anatomy who held the chair of anatomy at Edinburg University. Monro Secundus recorded detailed descriptions and illustrations regarding communication between lateral and third ventricle of the brain as well as describing changes seen in hydrocephalus [4].

Monro also made several important observations about cranial cavity with application of physical principles to the intracranial contents. George Kellie former student of Monro also studied about blood volume in human brains and reached the same conclusion as his mentor which is now known as Monro-Kellie hypothesis which states that the sum of volumes of brain parenchyma, CSF, and intracranial blood is constant [5].

2.3 Embryology

Central nervous system starts developing from fourth week of intrauterine life. Neural tube formed shows closure of anterior neuropore by middle of fourth week and posterior neuropore by end of fourth week. Cranial end of neural tube shows three dilated brain vesicles as prosencephalon [forebrain], mesencephalon [midbrain] and rhombencephalon [hindbrain]. The procesencephalon further subdivides into an anterior telencephalon which forms two cerebral hemisphere having



Figure 1.

A] Human embryo, approximately 10.2 mm long, left lateral surface of the diecephlon and telencephalon removed. B] Human embryo 13.6 mm long, median section. C] Human fetus, approximately 3 months old, median section showing medial surface of enlarged telencephalon and diencephalon with interventricular foramen of Monro. [Gray H. THE ANATOMICAL BASIS OF MEDICINE AND SURGERY. 40th ed. Susan Standring, Elsevier Churchill Livingstone New York; 2008. p.381–383.



Figure 2.

Sagittal section of brain showing following cerebrum, cerebellum and brainstem:A] frontal lobe, B] Paritel lobe, C] occipital lobe, D] cerebellum, E] corpus callosum, F] septum Pellucidum, G] fornix, H] thalamus, I] hypothalamus, J] pons, K] midbrain, L] medulla oblongata, black arrow- opening of interventricular foramen of Monro, yellow star – Interthalamic adhesion, black dotted line – Thalamohypothalamic sulcus.

lateral ventricle cavities and posterior diencephalon having third ventricle cavity. The commencement of cerebral diverticula from the wall of forebrain persists as interventricular foramen of Monro [6]. Ventricular cavities of brain develop in the neural tube and their enlargement depends of differential growth of the brain vesicles. Out of the three brain vesicles the cavity of the forebrain gives rise to two lateral and third ventricles. Lateral ventricle grows as outpouching from the rostral end of third ventricle and both are interconnected via foramen of Monro (**Figure 1**).

2.4 Gross anatomy

Foramen of Monro is small slit like communicating channel between paired lateral ventricle and third ventricle cavity on either side which become clinically significant when obstructed thus leading to non-communicating hydrocephalus.

Foramen of Monro is located at the junction of roof and anterior wall of lateral ventricle thus bounded anteriorly by the body and column of fornix and posteriorly by anterior nucleus of thalamus (**Figure 2**). Size and shape of the foramen correlates with that of ventricles. In an embryo it is large and circular and as the ventricle size increases, it narrows into a slit like opening. Not only choroid plexus but posterior choroidal, superior choroidal arteries, thalamostriate and septal veins also pass through it (**Figure 2**).

3. Neuro radiology

See Figure 3.

3.1 Spectrum of foramen of Monro lesions causing obstructive hydrocephalus (Table 1)

The 3rd ventricle is bounded by the interventricular foramen on either side where the roof and anterior wall of the third ventricle meet the body of the fornix along with the column anterior to the foramen. Posteriorly it is related to the thalamus [anterior pole].

Literature shows that tumors in the vicinity of the foramen are uncommon and usually cause hydrocephalus. Various pathologies which can lead to foramen of Monro obstruction and obstructive hydrocephalus include colloid cyst, craniopharyngioma, subependymal giant cell astrocytoma [SEGA], neurocysticercosis, tuberculous meningitis, pituitary macroadenoma, neurocytoma, ventriculitis, multiseptate hydrocephalus, intraventricular hemorrhage,



Figure 3. Normal interventricular foramen and its relations on MRI.

Types of hydrocephalus in foramen of Monro obstruction	Etiological classification	Pathological Classification
 Univentricular Biventricular Multiseptate hydrocephalus 	 Congenital Inflammatory Infective Neoplastic Traumatic/ hemorrhagic Functionally isolated ventricles 	 Lesional IIIrd Ventricle lesion Lateral ventricle lesion Septal lesion Extraventricular lesion Stenosis Septations/ membranes Adhesions/exudates Blood products

Table 1.

Classification of foramen of monro lesions causing obstructive hydrocephalus.

functionally isolated ventricles, choroid plexus tumors, subependymomas and idiopathic foramen of Monro stenosis.

4. Colloid cyst

4.1 Introduction

Colloid cysts are rare, congenital, histologically benign tumors which represent upto 2% of all intracranial neoplasms and correspond to approximately 15–20% of the intraventricular tumors [7]. They occur most commonly in the 3rd to 5th decades of life. They are usually solitary and sporadic, although rare examples of cysts on other locations and familial forms are known. They occur in the posterior end of the foramen of Monro in the anterior and antero-superior part of the third ventricle [8].

The colloid cyst is an epithelial lined cyst filled with gelatinous material which commonly contains mucin, old blood cholesterol and ions and may vary in size from 3 to 40 mm in diameter. However size of the cyst does not appear to be a reliable predictor of outcome, as death may occur even with smaller lesions [9]. Types of colloid material seen can be a] greenish liquid b] greenish liquid with cholesterol c] whitish colloid d] greenish colloid e] mixed.

The precise embryopathogenesis of colloid cyst is poorly understood and still a topic of debate. Various theories proposed are a] origin from either the diencephalic vesicle or the persistence of embryonic paraphysis b] derived from neuroepithelium c] remnant of respiratory epithelium and d] an ependymal cyst from the diencephalon [10].

Clinical presentation is heterogenous and may be intermittent, self- resolving and non-specific. Obstructive hydrocephalus is precipitated by growth of the colloid cyst blocking CSF flow through one or both foramen of Monro and may produce raised intracranial pressure [ICP] by intermittent obstruction of the passage of CSF at the level of interventricular foramen, acting like a legger of a ball as historically described by Dandy in 1933 [8].

4.2 Clinical features

Defining the natural history of colloid cyst reliably has been challenging due to the small number of cases in most case series. Majority of colloid cysts present as an

Cerebrospinal Fluid

incidental finding while imaging the brain for unrelated symptoms. Symptoms due to colloid cyst often result from different forms of hydrocephalus as well as irritation of major important centers around the third ventricle [11].

They do not have any intrinsic pathological properties and cause symptoms by acting as inert masses. 90% are asymptomatic and stable, while 10% are found to increase in size and cause hydrocephalus. Sudden increase in size can lead to drop attacks, dementia, coma, and death.

Headaches are the most common symptom of colloid cyst ranging from 65–100% of cases according to literature. The headaches are typically severe and intense, throbbing or aching in quality and can be bifrontal or generalized in location and can be precipitated, aggrevated or relieved by head movement or position changes. With progression in size of the cyst, the headaches become more frequent and can be accompanied frequently by nausea, vomiting, blurred vision,



Figure 4.

Case of a small colloid cyst presenting with drop attacks [A-D]: A] pre-op CT showing a hyperdense SOL at foramen of Monro. B] Endoscopic view of the ventricular anatomy and colloid cyst. C] Normal ependyma with 3rd ventricular floor post excision. D] Post op CT showing complete excision of lesion with re-established CSF flow across foramen of monro.

gait ataxia, cognitive decline and less frequently by dizziness, tinnitus and dysautonomic symptoms like abdominal pain, tachycardia, hyperthermia, bradycardia and sweating. Other uncommon symptoms include personality changes, memory disorders, psychiatric disorders and olfactory and gustatory hallucinations. Rapid deterioration due to acute hydrocephalus can occur in 3–35% of patients with an associated 5–38% risk of death [11–13].

4.3 Neuroradiology

Due to the different composition and density of contents, which depends on the quantity of cholesterol and proteins, colloid cysts may have a diverse appearance on imaging. Cysts with high cholesterol and protein content are hyperdense on plain CT, hyperintense on T1 and hypointense on T2 weighted images.

Computed Tomography [CT]: On CT, the colloid cyst is typically a well-defined, round or oval hyperdense mass in the anterior third ventricle at the foramen of Monro. Precontrast scans show a] hyperdense lesions in approximately 2/3rd cases, b] isodense in 1/3rd cases and c] rarely can be hypondense or show calcifications. (**Figure 4**).

On post contrast CT, colloid cysts usually do not show enhancement. Less commonly they may show mild to moderate contrast enhancement. In some cases, thin rim of contrast enhancement may be present which is thought to represent the cyst capsule [8].



Figure 5.

case of a large colloid cyst presenting with signs of raised ICP A] & B] preoperative Sagittal T1 and Coronal T2 images isodense third ventricular lesion extending into the septum pellucidum. C] preoperative contrast enhanced axial image D] intraoperative image with whitish colloid material being excised E] & F] postoperative T1 sagittal and T2 coronal images showing near total excision.

4.4 Magnetic Resonance Imaging

MRI is the investigation of choice for imaging colloid cysts. On MR imaging, some lesions may show intracystic fluid levels or central and peripheral components in the lesions whereas some lesions are homogenous in appearance.

On T1 Sequencing, colloid cyst is homogenously hyperintense in about 50% of cases while the other lesions can be hyperintense, isointense or hypointense. On T2 Sequence imaging, most colloid cysts are hypointense. Low signal intensity on T2 imaging may correspond to the viscous contents of the colloid cyst and thus harder to aspirate. Post contrast enhancement of the cyst is not seen usually and rarely a peripheral enhancement will be noted around the cyst which represents a vessel stretched over the colloid cyst. (**Figure 5**)

Colloid cysts usually have a similar intensity to surrounding CSF on FLAIR sequencing and may have decreased signal intensity on diffusion weighted imaging [10].

4.5 Treatment

For patients who are symptomatic and have a higher degree of ventriculomegaly, more immediate surgical options include craniotomy for microsurgical resection, neuroendoscopic removal, and CSF -diversion with ventriculo-peritoneal [VP] shunts.

The transcallosal microsurgery and endoscopic approach were compared for the first time by Lewis et al. in 1994. The findings of their comparison were shorter stay in the hospital and early recovery in the endoscopic approach group [14].

In a study by N.B. Levine et al. from 1991 to 2004, the conclusion was made that endoscopy approach should be offered to all patients as the first line of management [15].

Criterion	Points
Age < 65 years	
Yes	1
No	0
Headache	
Yes	
No	0
Axial diameter ≥ 7 mm	
Yes	1
No	0
FLAIR hyperintensity	
Yes	1
No	0
Risk zone	
Yes	1
No	0

Table 2.Colloid cyst risk score.

In 2016, Beaumont et al. published the Colloid Cyst Risk Score [CCRS], a method used to stratify the risk of a patient to develop obstructive hydrocephalus and guide physicians to choose appropriate treatment pathways. Risk of lesion progression and obstructive hydrocephalus is more with a CCRS \geq 4 [13] (**Table 2**).

In 2016, Suresh Nair et al. published a study which was done from 1980 to 2011, 275 cases were managed by interhemispheric transcallosal approach, twenty-two by transcortical approach and 8 by endoscopic approach. They concluded that open microsurgical approach is the gold standard surgical treatment for colloid cyst [16].

Author recommendations: Neuroendoscopic approach can be offered as the first line treatment for colloid cyst as it can be used for all classic colloid cysts irrespective of the cyst site.

The entry burr hole should be 1 cm lateral to the usual Kocher's point to facilitate endoscopic septostomy. Depending on the intraoperative bleeding, consistency of the cyst and the extent of resection, the decision to place a post-operative EVD can be customized, which can be removed after 4–5 days. Rarely, VP shunt might be needed in these cases.

Case illustrations (Figures 4 and 5).

5. Craniopharyngioma

5.1 Introduction

Craniopharyngioma [CP] is a benign tumor originating from the squamous epithelial residual cells anywhere along the obscured craniopharyngeal duct from Rathke's cleft to the floor of the third ventricle and they constitute 2–4% of intracranial neoplasms [17]. They often present a surgical challenge to the neurosurgeons because of their central location and proximity to surrounding neural structures namely the hypothalamus, pituitary gland, the optic apparatus, circle of Willis, brainstem and temporal lobes.

Pathologically these tumors are classified into two types: adamantinomous CP [ACP] and papillary CP [PCP]. ACPs are more common than PCPs, are pathologically distinct, are composed of cystic "motor oil-like" component and solid component with frequent calcifications and are more common in pediatric population while PCPs are more common in the adult population [18].

The most common location of craniopharyngioma is the sellar and suprasellar region with 95% of the tumors having a suprasellar component. The suprasellar component of the tumor grows in various directions compressing the surrounding structures. The anatomical proximity of the tumors to the major CSF pathways may result in compression of various parts of the ventricular system causing obstructive hydrocephalus. Obstruction in these cases can be seen at the following levels a] basal cisterns b] invasion and obstruction of the inlet and outlet of the third ventricle c] foramen of Monro and rarely d] posterior displacement of the brainstem with occlusion of the Sylvian aqueduct [19].

Kassam et al. classified suprasellar craniopharyngiomas on the basis of relationship of the tumor to the infundibulum and pituitary stalk as observed in the surgical field on endoscopic viewing which is as follows: I] pre-infundibulum: Its lateral relation are carotid arteries, below by the diaphragm sella, infundibulum in the dorsal portion, and displaced chiasm in the roof. II] trans-infundibulum: The rostral extent of the tumor is bounded by the anterior portion of the hypothalamus. III] retro-infundibulum: a] Extending into the third ventricle. b] Extending into the inter-peduncular cistern. It is bounded anteriorly by the stalk and posteriorly by the mammillary bodies and basilar apex. IV] intra-ventricular and/or optic recess [20]. Generally Kassam's type III and type IV tumors present with obstruction at the level of foramen of Monro.

5.2 Clinical features

Clinical manifestations are related to hypothalamic and pituitary deficiencies, visual impairment and raised intracranial pressure [17].

Headaches are seen in about 50% of patients which may be due to raised intracranial pressure or due to meningeal irritation from the cystic fluid and can be associated with nausea and vomiting.

Symptoms of endocrine dysfunction are seen in 52–87% of patients which are caused by tumor or treatment related lesions to the hypothalamic–pituitary axis that affect the secretion of growth hormone [GH] in about 75% patients, gonadotropins [FSH/LH] in about 40% patients and adrenocorticotropic hormone [ACTH] in about 25% patients. Patients can also present with vasopressin deficiency causing diabetes insipidus in about 20% of cases.

5.3 Investigations

Evaluation and management of craniopharyngioma requires an interdisciplinary approach by endocrinologist, neuro-ophthalmologist and neurosurgeon.

5.4 Neuroradiology

Both CT and MRI are helpful for diagnosis of craniopharyngiomas which are heterogenous tumors with solid, cystic and calcified components.. PCPs are usually solid tumors with rare cystic transformations and calcifications. Radiological features of ACPs can be summarized by the 90% rule i.e. 90% of tumors are predominantly cystic, 90% show more or less prominent calcifications and about 90% take up contrast in the cyst wall [21].

CT provides details of the sellar anatomy as well as information related to cystic and solid components of the tumor, local invasion, compression of adjacent structures and calcifications.

MRI is also useful for the topographic and structural evaluation of craniopharyngiomas. These tumors are isointense or hypointense on T1 weighted images, hypointense or hyperintense on T2 weighted images and show contrast enhancement. This variability in MRI findings are due to varying proportions of solid and cystic components and presence of cholesterol, keratin, hemorrhage and amount of calcification.

5.5 Treatment

The choice of the surgery depends on the anatomical location of the tumor. It has been reported that gross total resection causes more neurological deficits and does not improve the chances of its recurrence. The Endoscopic approach is not recommended for very large tumors with solid component, calcifications or vascular invasion. Kassam's type I and type II tumors can be approached by endoscopic transnasal transsphenoidal technique. However for Kassam's type III and type IV tumors have to be approached by endoscopic transventricular or transcranial approaches (**Table 3**).

The decision regarding the approach to the lesion is guided by the following factors a] consistency of the lesion b] calcification c] lateral and extraventricular extension of the tumor.



Table 3.

Algorithm for surgical approaches for predominantly third ventricular craniopharygiomas.

Authors recommendations: In the event of endoscopic transventricular approach for craniophryngiomas, the authors strongly recommend endoscopic septostomy and post operative EVD placement in all the cases. VP shunt may be required for permanent CSF drainage depending on the extent of resection. The incidence of postoperative VP shunt at a later date is relatively more common in craniopharyngiomas than colloid cyst. Also Omaya reservoir can be placed post operatively for intralesional bleomycin in predominantly cystic lesions limited to the third ventricle, although the authors have a limited experience regarding the same.

In a study by Deopujari et al. from 2000 to 2016 it was stated that suprasellar craniopharyngiomas with a major cystic component can be best managed by a combined endoscopic transcranial and transnasal approach [22].

Case illustration (Figure 6).

The toxic radioactive substances like bleomycin, interferon alpha can be given in craniopharyngiomas with cystic component only to promote sclerosis and fibrosis. One of the major disadvantages of this approach is that it can produce severe neurotoxicity and leakage of the sclerosing substance [23].

For cystic craniopharyngiomas radioisotopes can be implanted through an endoscopic approach. However, there are no standard guidelines for the intra cystic dosage and the toxicity level of these drugs along with tumor control dosage, all these makes this therapeutic option difficult to follow [24].



Figure 6.

case of craniopharyngioma [Kassam's type III] presenting with dwarfism and raised ICP A] pre-operative non contrast axial CT B], C] \mathcal{C} D] pre-operative contrast enhanced axial, coronal and sagittal CT images respectively E] \mathcal{C} F] postoperative non contrast axial and sagittal CT image.

6. Subependymal Giant Cell Astrocytoma [SEGA]

6.1 Introduction

SEGA also known as Subependymal Giant Cell Tumor [SGCT] are clinically benign [WHO grade 1], slow growing tumors which usually arise in the periventricular regions in the proximity of the foramen of Monro. They are associated with tuberous sclerosis complex [TSC], which is a systemic autosomal disease characterized by Vogt's clinical triad of mental retardation, seizures and facial angiofibroma. TSC has a prevalence ranging from 5 to 20% with radiographic evidence of subependymal nodules which are a precursor of SEGA seen in 90% of TSC patients [25, 26].

These are histologically benign tumors and do not undergo malignant transformation. The lesions which are over 10 mm in diameter at the foramen of Monro can cause obstruction of CSF flow leading to progressive dilatation of the lateral ventricles and raised ICP and cause the clinical manifestations associated with SEGA [25].

6.2 Clinical features

Preoperative diagnosis of SEGA takes into account the age, clinical condition of the patients and the location of the tumor. In cases where neurocutaneous manifestations of TSC which include mental retardation, seizures and adenoma sebacum [Vogt's triad] are present, early diagnosis of TSC is possible. However solitary

lesions without clinical or radiological features of TSC have also been reported and these patients almost always present urgently due to raised ICP.

In the early stage, patients can present with insidious onset subtle behavior changes, cognitive impairment or seizures. Features of raised ICP like headache and vomiting are present due to increase in size of the lesion causing obstructive hydrocephalus or due to intratumoural or intraventricular hemorrhage [26, 27].

6.3 Investigations

CT and MRI characteristics in SEGA are usually nonspecific. Patient factors which include age and location of the lesion are useful indicators in establishing diagnosis. Although nonspecific, radiological findings show a well circumscribed lesion at the foramen of Monro which is isodense or slightly hyperdense on CT with rare thin calcifications, hypointense on T1, hyperintense on T2 with marked contrast enhancement [26, 28].

6.4 Treatment

SEGAs are considered to be benign lesions and excision of the tumor is considered to be curative in patients presenting with a single lesion. Surgical treatment is indicated in cases of symptomatic SEGA or patients presenting with acute increase



Figure 7.

Young male patient with cutaneous manifestations of TSC presented with sudden onset altered sensorium A] Preoperative CT brain a circumscribed lesion in the right foramen of Monro region with calcification, hemorrhage and obstructive hydrocephalus B] Preoperative MRI showing heteregenous enhancement with mixed cystic and solid areas and multi-stage hemorrhages C] Clinical picture showing cutaneous manifestations of Tuberous Sclerosis Complex D] Follow up CT brain at 2 years showing near total excision of the tumor except the calcified part with resolution of hydrocephalus.



Figure 8.

20 year old female with cuteneous manifestations of TCS presenting with raised ICPA]& B] preoperative MRI showing a contrast enhancing solid lesion at the level of right foramen of Monro leading to univentricular [right lateral ventricle] hydrocephalus. C] & D] endoscopic septostomy was done to deal with the obstructed right ventricle with endoscopic biopsy which was suggestive of SEGA. E] & F] postoperative imaging showing septostomy defect. Later patient was managed with m-TOR inhibitors [rapamycin] with progression free survival over a period of 2 year follow up.

in intracranial pressure due to obstructive hydrocephalus [29]. Other alternative treatment options are Gamma Knife radiosurgery [GKR] or the mechanistic target of rapamycin [mTOR] inhibitors which can reduce the size of the mass in TSC. GKR showed promising results for many types of benign brain tumors, including gliomas, with a low incidence of side effects [30].

Authors recommendations: In our experience, SEGA's were operated by open transcallosal/ transventricular approaches to the lateral ventricle. As these tumors are generally solid and firm in consistency, they are not amenable to endoscopic excision.

Case Illustrations (Figures 7–9).

7. Neurocysticerosis

7.1 Introduction

Neurocysticercosis [NCC] occurs when larval stage of the tapeworm Taenia Solium migrates to the central nervous system. It is the most common helminthic infestation of the central nervous system and usually manifests as acute seizure, epilepsy, progressively worsening headache or focal deficit [31].

The cysts reach the ventricular system through the choroid plexus and are more frequently found in the fourth ventricle. This can be attributed to the gravitational



Figure 9.

Mentally retarded child with neurocutaneous markers of TSC presented with raised ICP Figure A], B] \mathfrak{G} C] MRI revealing large solid enhancing lesion with calcifications at the level of Foramen of monro extending into the lateral ventricle, third ventricle and involving the septum pellucidum causing obstructive hydrocephalus. D], E] \mathfrak{G} F] Post- operative image showing near total excision with VP shunt in situ [which was done at a later date].

forces which favor migration from supratentorial ventricles or the cysts may directly enter through the choroid plexus [32].

The locations of intraventricular cysts determine the natural history of the disease. Ones attached to the ventricular wall involutes and eventually resolve, while the cysts which are not attached may migrate and block the cerebrospinal flow causing obstructive hydrocephalus. Hydrocephalus can occur either due to ventricular obstruction or arachnoiditis. Sites of obstruction include foramen of Monro, third ventricle, aqueduct of Sylvius and fourth ventricle [32, 33].

7.2 Clinical features

Active intraventricular cysts may remain asymptomatic for years and may become symptomatic if they obstruct the CSF flow leading to hydrocephalus and raised ICP. Abrupt obstruction may lead to acute hydrocephalus and present with features of hydrocephalus which include headache, nausea, vomiting, diplopia, restlessness, seizures, respiratory changes, bradycardia, hypertension, altered sensorium and papilledema or may present infrequently with features of Brun's syndrome [34].

Death of an intraventricular cyst larva can lead to liberation of antigenic substances being released into the ventricular system with local reactions causing an inflammatory response throughout the ventricular system. These patients will present with features of raised ICP, meningoencephalitis, focal neurological deficits and inflammatory reaction detectable in CSF [35].

7.3 Investigations

The diagnosis of intraventricular NCC is based on systematic clinical evaluation, neuroimaging and serology. However the role of serology is limited due to low sensitivity and specificity. Various serological tests include a] antibody testing with immunoblot assay using T. solium antigens on serum or CSF samples b] direct antigen testing of Taenia solium antigen with ELISA using a monoclonal antibody against Taenia saginata HP10 antigens on CSF or serum samples c] detection of Taenia solium specific DNA in CSF by polymerase chain reaction [36].

7.4 Neuroradiology

Identification of scolex in a cystic lesion is the pathognomonic radiological finding in NCC [32, 34, 35]. Non-contrast CT is sensitive for parenchymal and calcified lesions but is not sensitive for extraparenchymal disease. CT may fail to demonstrate small cysts that do not deform the ventricles as a] they share the same density of CSF b] the cyst wall and the scolex are not visible c] the cyst does not show contrast enhancement.

MRI is the investigation of choice for extraparenchymal NCC as the MRI properties of the scolex or the cystic fluid differ and inflammation is marked with hyperintensity signals which allow a reliable diagnosis.

A viable active intraventricular cyst appears as a well defined thin walled cystic lesion of 10–20 mm in diameter which is hypointense on T1 with a thin rim of hyperintensity. The scolex is seen as an eccentric rounded or elongated enhancing mural nodule of 2-4 mm in diameter within the cyst cavity. On T2 weighted imaging, the contents of the cyst are isointense with the surrounding tissues with an hyperintense scolex. Contrast enhanced T1 weighted MRI imaging shows contrast ring enhancement of the cysts with surrounding edema.

7.5 Treatment

Treatment modalities include antiparasitic drugs, surgery and symptomatic medications. Medical management includes a] corticosteroids for meningitis, cysticercal encephalitis and angitis b] antiparasitic drugs which include praziquantel and albendazole [36].

Principles of surgery include treatment of hydrocephalus and removal of cyst. Modalities of surgical intervention include a] emergency ventriculostomy b] placement of VP shunt c] Neuroendoscopic or microsurgical extirpation of obstructing cysts.

As these cysts are not densely adherent to the ventricular wall endoscopic approach is the preferred surgical option. There is usually no enhancement of the cyst wall but in cases where the cyst wall is enhancing, it is suggestive of either ependymal inflammation or adherence of cyst wall to the ependyma. Thus, the decision to approach these lesions endoscopically, in cases of enhancing cyst wall, should be taken with caution. Psarros et al. in his study made an observation that despite rupture of the cyst and spillage of contents in the ventricle, ventriculitis was not seen. Continuous perioperative irrigation with ringers solution helps in removing the debris and provides clear vision [37].

Authors recommendations: Post operative External ventricular drain [EVD] should be placed to address the spillage of contents in the ventricle, but however in our experience we did not find any inflammatory reaction because of the cyst contents.

Case Illustration (Figure 10).



Figure 10.

12 year female presented with Bruns syndrome [episodic symptoms of raised intracranial pressure]. A], B] \mathfrak{G} C] MRI images showing a cystic lesion in the left lateral ventricle obstructing the left foramen of Monro leading to obstructive hydrocephalus and deviation of the septum pellucidum to the right. D] \mathfrak{G} E] intraoperative photo showing the excision of the white cyst wall with complete decompression of the left foramen of Monro. F] Postoperative image showing complete excision with midline septum pellucidum.

8. Tubercular meningitis

8.1 Introduction

TBM, a frequent form of central nervous system tuberculosis is a serious neurological disease with significant mortality and morbidity. In India, the estimated mortality due to TBM is approximately 1.5/100,000 population. Neurological complications like cerebral infarctions and hydrocephalus are common and may cause worsening of prognosis [38].

Hydrocephalus, one of the most common complications of TBM, can occur early or late in the clinical course and can also be associated with the commencement of anti-tubercular drugs.

Hydrocephalus in TBM could be either of communicating or obstructive type, the former being more common. The main cause of hydrocephalus in both types is by presence of thick basilar exudates in the subarachnoid spaces or the ventricular pathways [39].

Obstructive hydrocephalus results from block or compression within the fourth ventricle due to exudates or leptomeningeal scar tissue or obstruction of aqueduct of Sylvius due to strangulation of brainstem by exudates or subependymal tuberculoma [40].

Foramen of Monro is anatomically narrowed by the bulk of the choroid plexus which is susceptible to obstruction due to a] meningeal inflammation leading to raised CSF protein content rendering the CSF more viscous and compromising CSF flow b] focal ventriculitis c] exudates/ scarring in the region of foramen of monro [41].

8.2 Clinical features

Progression of disease in patients with obstructive hydrocephalus in TBM is generally rapid as compared to those presenting with communicating hydrocephalus [42].

In addition to the primary and constitutional symptoms of tubercular meningitis, obstructive hydrocephalus should be suspected in any patient with TBM presenting with sudden onset altered sensorium with or without presence of papilledema or in patients complaining of rapidly progressive headache with or without blurring of vision.

8.3 Neuroradiology

Contrast enhanced CT and MRI are helpful in diagnosing the complications of TBM which include presence of hydrocephalus, subependymal seepage, tuberculomas infarcts, edema, nodular enhancing lesions and basal exudates [40].

8.4 Treatment

Medical management includes: antituberculous therapy, steroids, dehydrating agents like mannitol and diuretics such as frusemide and acetazolamide to reduce CSF production [40, 43].

Surgical management includes CSF diversion surgery which can be done conventionally with a VP shunt or neuroendoscopically.

The advantages of neuroendoscopy are:

- a. endoscopic septostomy and monroplasty can be done which obviates the need of biventricular shunt
- b. provides biopsy sample for further confirmation of the diagnosis



Figure 11.

A] Intra-operative image showing obstruction at the level of foramen of Monro with ependymal tubercles B] perforation of the septum followed by dilatation with C] balloon catheter and D] dilating forceps E] post foraminoplasty restoration of caliber of the foramen of monro with no damage to surrounding structures.

- c. adhesiolysis can be performed at the level of foramen of monro
- d. clearance of exudates decreases the bulk of the infection and restores the CSF flow which in turn reduces the convalescence period

In a study by Chugh et al. [43]. it was concluded that in cases of TBM with hydrocephalus endoscopic procedures can be offered as the first line of management as it provides the added advantage of performing septostomy, monroplasty and biopsy at the same sitting.

Case Illustration (Figure 11).

9. Pituitary adenoma

9.1 Introduction

Pituitary tumors are a heterogenous group of CNS lesions that are usually benign and constitute 10–15% of all primary intracranial tumors in adults. They can be divided on the basis of their size into macradenomas [diameter more than 10 mm] and microadenomas [size less than 10 mm. Nonfunctioning pituitary adenomas with diameter exceeding 40 mm are considered as giant adenomas. These adenomas are rare and frequently invade the suprasellar structures causing mass effect [44, 45].

Obstructive hydrocephalus due to a pituitary adenoma is rare with very less literature available, limited to case reports. Hydrocephalus is rarely the presenting symptom or can occur during the course of the disease.

Various mechanisms of development of hydrocephalus in pituitary adenomas include: a] due to lesser resistance, most pituitary macroadenomas have cranial and anterior extension. The tumor grows along the pituitary stalk upwards through the sellar diaphragm to impinge on the recesses of the third ventricle. In rare cases, the tumor becomes large enough to obstruct the intraventricular foramen leading to secondary hydrocephalus. b] some tumors extend along the floor of the third ventricle and obstruct the Sylvian aqueduct causing obstructive hydrocephalus. c] through obliteration of suprasellar cistern [46].

9.2 Clinical features

Clinical manifestations of giant pituitary adenomas can be secondary to compression of the surrounding structures, pituitary hormone deficiency or hypopituitarism and tumor hypersecretion [44, 46, 47]. Common manifestations of non functioning pituitary adenomas include headache, visual impairment and visual field defects. Obstructive hydrocephalus is characterized by symptoms of raised ICP which include headache, nausea, vomiting, blurring of vision, memory loss, irritability, personality changes, papilledema, sleep disturbances, gait disturbances, loss of bladder control and coma. Involvement of the frontal lobes can be associated with generalized seizures and dementia. Extension of the tumor into the cavernous sinus can be associated with third, fourth and sixth nerve palsy.

9.3 Investigations

Necessary investigations for diagnosing a pituitary tumor include hormonal profile, neuroradiology and fundoscopy.

9.4 Neuroradiology

Contrast enhanced MRI is the investigation of choice for size and location of the tumor and its relation to surrounding structures [48]. CT can provide additional information which include identification of bone destruction and confirmation of suspected intra/parasellar calcifications (**Figure 12**).



Figure 12.

60 years old male presented with gradual diminution of vision with altered sensorium with A], B] C C] preoperative scans showing a sellar, suprasellar and right parasellar lesion [pituitary adenoma] with a cystic lesion superior to the adenoma causing right foramen of Monro obstruction. D] Intraoperative picture during transnasal transsphenoidal showing complete decompression of the sellar and suprasellar part of the lesion with the diaphragm Sella herniating in the Sella. E], F] postoperative CT showing decompression of the sellar lesion but persistence of the cystic lesion in the right lateral ventricle. G] Neuroendoscopic exploration of the right ventricle shows cystic lesion with xanthochromic fluid with underlying foramen of Monro. H] Post cyst decompression, foraminoplasty being performed. I] Rest of the lateral ventricle examined for remnants of cyst. Also a septostomy was performed and Ommaya reservoir placed post operatively. J], K] \Huge{C} L] post operative scan revealing adequate decompression of the level of foramen of Monro.

9.5 Management

The goals of surgery include tumor removal, relieving mass effect, improving visual abnormalities, reducing hormone hypersecretion to normal levels and preserving pituitary function [44, 46].

The various modalities of treatment include

a] transsphenoidal approach b] transcranial approach c] combined or two stage approach

In a study by B.K.Ojha et al. it was documented that, a combined transsphenoidal and simultaneous transventricular/endoscopic approach is a safe and effective option for giant pituitary macroadenomas in which tumor dimensions, consistency or history of previous surgical treatment were indicative of incomplete removal by single approach alone [49].

Case Illustration.

10. Intraventricular hemorrhage

10.1 Introduction

Intraventricular hemorrhage [IVH] are classified as a] primary – involving the ventricular system and adjacent ventricular lining without associated parenchymal or subarachnoid hemorrhage which occurs in about 30% of cases. b] secondary-primary intracerebral hemorrhage [ICH] or subarachnoid hemorrhage [SAH] extending into the ventricular system which occurs in about 70% of patients and is an independent predictor of poor outcome [50].

Pathophysiology of obstructive hydrocephalus in IVH is due to the blood clot blockage in the CSF pathway. Other contributory factors causing hydrocephalus in these cases include a] release of inflammatory mediators by the blood components causing a secondary response b] damage to the ependymal cells lining the ventricles due to inflammation c] fibrosis and scarring of the arachnoid granulations d] complement activation [51].

10.2 Neuroradiology

CT is the investigation of choice for diagnosing IVH with or without ICH as it allows for rapid diagnosis and prompt management (**Figure 13**). Blood is easily identified on CT as a white hyperdense lesion. CT is also useful to identify other important factors such as edema and hydrocephalus. Pattern and topography of bleeding can give important clues about the secondary causes of IVH and additionally CT angiography or contrast enhanced CT can be done for the same [52].

MRI is equally effective to identify acute hemorrhage. It is useful to distinguish between hemorrhage and an ischemic stroke, although it would be a less preferable investigation in an emergency situation [53].

10.3 Treatment

Goal of treatment is to limit hemorrhagic mass effect, edema, obstructive hydrocephalus by rapid removal of blood and blood products from the ventricular system [54]. Modalities of treatment in these cases include a] EVD insertion b] EVD combined with use of thrombolytics c] Neuroendoscopic aspiration.

Case Illustration (Figure 13).



Figure 13. *CT scan A: showing IVH with dilated ventricles. B: Showing EVD in-situ with resolution of IVH and hydrocephalus.*

11. Ventriculitis

11.1 Introduction

Nosocomial infection of the central nervous system are a serious complication of patients undergoing neurosurgical procedures like craniotomy, placement of invasive neuromonitoring technique, EVD catheters or CSF shunts.

EVD, which is a common procedure for controlling and monitoring raised ICP secondary to acute occlusive hydrocephalus has an associated major complication of bacterial colonization of the catheter and subsequent retrograde infection resulting in encephalitis, ventriculitis, meningitis, brain abscess, subdural empyema or even sepsis [55].

Ventriculitis, also termed as ependymitis, ventricular empyema, pyocephalus, ventricular abscess or pyogenic ventriculitis, is the inflammation of the ependymal lining of the cerebral ventricles. It can be secondary to meningitis, cerebral abscess, EVD or shunt related, trauma, CSF leak or as a complication of intrathecal chemotherapy.

Incidence of ventriculitis according to literature ranges from 5–20% and incidence of ventricular catheter related ventriculitis ranges from 5–45% [56].

Most common pathogens causing ventriculitis are skin flora which include *staphylococcus epidermidis* [70%] and *staphylococcus aureus* [10%]. Other less common pathogens include gram negative rods [Klebsiella spp., *E. Coli*, Pseudomonas spp], anaerobes and candida spp. [56].

Obstructive hydrocephalus in these cases is due to the obstruction caused by the exudates, adhesions and septations at the level of foramen of Monro or the aqueduct.

11.2 Clinical features

Patients can present with headache, nausea and vomiting, fever, altered mental state, meningism, focal neurological deficits and features of secondary hydrocephalus and raised ICP. On local examination over the subcutaneous shunt tubing, erythema or tenderness can be seen suggestive of infection [57].

11.3 Investigations

Diagnosis of ventriculitis is done by CSF examination and neuroradiology.

11.4 Neuroradiology

Ultrasound can be performed for neonates by using a high-frequency transducer through the anterior fontanelle in coronal and sagittal planes. The most common findings include an irregular and echogenic ependyma, the presence of intraven-tricular debris and stranding, intraventricular adhesions and septae associated with ventricular dilatation [58].

Noncontrast CT may be nonspecific which include dependent hyperdense ventricular debris, univentricular or biventricular hydrocephalus, periventricular low density and features of underlying abnormality. Contrast enhanced CT shows homogenous enhancement of the ependymal lining of the ventricles (**Figure 14**).

MRI findings are similar to CT, with the ventricular debris hyperintense T1 weighted images and hypointense on T2 weighted images, with high signal on DWI, reduced ADC value and are seen as fluid levels of high signal intensity on FLAIR images [57].

11.5 Treatment

On clinical suspicion of nosocomial ventriculitis, empiric antibiotic therapy should be initiated.

Surgical modalities for ventriculitis include a] intraventricular/intrathecal antibiotics b] continuous intraventricular irrigation therapy using a closed drainage system c] neuroendoscopic ventricular lavage with septostomy/ monroplasty [59].

Rational for early surgery in ventriculitis: The rapid development of polydrugresistant class of gram-negative bacteria, poor passage of antibiotics in the intraventricular space after intravenous injection and hampered CSF flow from ventricles along with infection cause the ventricles to become an enclosed system in



Figure 14

5 year female child presented with A] monoventricular [left lateral ventricle] hydrocephalus post EVD insertion. B] Intraoperative evidence of exudates causing blockage of left foramen of Monro. C] Intraventricular exudates were cleared. D] Foraminoplasty done with E] Septostomy. As there was evidence of exudates in the right lateral ventricle which could be seen from the septostomy, neuroendoscopic lavage was given bilaterally. EVD was inserted on the left side. F] Immediate postoperative radiology showing bilaterally equal ventricles with resolving hydrocephalus.

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which infected CSF collects and the low concentration of antibiotics after intravenous injection is insufficient to kill the bacteria [60].

The endoscope allows seeing the entire cavity of the ventricles and also facilitating endoscopic lavage and clearance of the infected CSF. The decrease in infective load increases the effectiveness of the intrathecal antibiotics. Lavage inside the ventricles with continuous irrigation over a prolonged period has been reported with favorable outcomes.

Authors recommendations: We are of the opinion that in cases of ventriculitis a biventricular lavage with removal of ventricular exudates and adhesions is better than dealing the infection through one ventricle only.

Case Illustration (Figure 14).

12. Central neurocytoma

12.1 Introduction

Central neurocytoma is a rare benign tumor of neuronal differentiation that is classified as grade II by World Health Organization [WHO]. They comprise about 0.1 to 0.5% of all brain tumor and are typically seen in young adults around the third decade. They are characteristically located in the supratentorial ventricular system more commonly involving the foramen of Monro and in lesser frequency in the third and fourth ventricle [61].

12.2 Clinical features

These patients present with signs and symptoms of raised ICP induced by obstructive hydrocephalus [61, 62]. Typical clinical symptoms include headache, nausea, vomiting, seizures, paresthesias, balance problems, decreased consciousness, weakness, memory and visual disturbances. In rare cases, IVH may also occur.

12.3 Neuroradiology

On noncontrast CT, central neurocytoma appears hyperdense with calcifications seen in around 50% of cases which are usually punctate in nature [62, 63]. Cystic degeneration, seen usually in larger tumors can lead to heterogenous appearance of the tumor. CNs have mild to moderate enhancement on contrast enhanced CT. On MRI, CNs are hypo to isointense on T1, iso to hyperintense on T2, with moderate contrast enhancement (**Figure 15**).

12.4 Treatment

Surgical management with gross total resection is the treatment of choice [62, 64]. Goals of surgery are a] to establish the CSF pathway, determine the histopathological diagnosis and establish maximal surgical resection with minimum risk of neurological impairment. Radiotherapy to the tumor bed is debatable and is indicated in cases with incomplete resection to prevent tumor progression and recurrence. Stereotactic radiosurgery has also been proved to be effective compared to radiotherapy in terms of diminishing tumor recurrences and radiation associated complications in these cases.

In a study by Chun li et al. on 9 cases of central neurocytoma 2 patients died in the postoperative period. They concluded that the possibility of recurrence of



Figure 15.

30 year male patient presented with features of raised ICP with MRI findings suggestive of a lesion arising from the septum pellucidum causing obstruction at the level of right foramen of Monro. Endoscopic surgery was planned to decompress the tumor with biopsy. However owing to intraoperative intratumoral hemorrhage, a decompressive craniectomy had to be performed and the tumor was decompressed through transcortical route. Biopsy was suggestive of central neurocytoma [WHO grade 2] with MIB1-Labeling index following which patient was referred for radiotherapy.

central neurocytomas should be considered based on the histologic features, especially proliferation index [MIB1-LI] [65].

Case Illustration (Figure 15).

13. Functionally isolated ventricles

In some cases, rapid therapeutic drainage of one lateral ventricle particularly after a low pressure VP shunt, can cause an ipsilateral slit ventricle which functionally obstructs the foramen of Monro [66]. This functional obstruction may lead to dilatation of the contralateral ventricle.

The treatment of choice of functional ventriculomegaly is neuroendoscopic intervention with septostomy with third ventriculostomy (**Figure 16**).

Case Illustration (Figure 16).



Figure 16.

34 year male, recently shunted for TBM with hydrocephalus was referred with altered sensorium. MRI brain was suggestive VP shunt in situ in right lateral ventricle and asymmetric dilatation of the left lateral ventricle. Neuroendoscopy did not reveal any obstruction at the foramen of monro and hence septostomy was done following which patient improved.

14. Subependymomas

14.1 Introduction

Subependymomas are rare, benign, indolent tumors of ependymal origin which comprise of 0.2%–0.7% of all intracranial tumors. They are slow growing neoplasms frequently seen in middle aged men more commonly in the fourth [50–60%] and the lateral [30–40%] ventricles. They also have a predilection for the spine and are seen in the cervical and cervicothoracic region [67, 68].

These tumors are frequently asymptomatic found incidentally on autopsy or on neuroimaging done for other medical indications.

14.2 Clinical features.

Symptomatic presentation depends on the location and size of the lesion. According to literature, tumors located at the septum pellucidum and the foramen of Monro with a size of more than 4 cms were more likely to become symptomatic with signs of raised ICP. Cases with spontaneous intratumoral hemorrhage have also been reported causing obstructive hydrocephalus. Despite the benign nature of the tumor, cases of tumor recurrence and CNS metastasis have also been reported [69, 70].

14.3 Neuroradiology

Radiological appearance of these tumors can vary depending on their location. On MRI, these tumors are typically well demarcated nodular lesions typically in the fourth or lateral ventricle which are hypo to isointense on T1 weighted images, hyperintense on T2 weighted images with minimum contrast enhancement [68, 69].

14.4 Treatment

Total surgical resection is the treatment of choice as these tumors are well demarcated, noninvasive and avascular. Role of postoperative radiation is debatable and is reserved in cases with subtotal surgical resection or in case of recurrence [71, 72].

15. Idiopathic Foramen of Monro stenosis

Idiopathic stenosis of foramen of Monro is a rare cause of enlargement of the lateral ventricles. It can occur due to an absent or stenosed foramen of Monro or when a membrane occludes a normal sized foramen of Monro. Patients are usually asymptomatic but can occasionally present with symptoms of raised ICP [71, 73].

Contrast enhanced MRI is the investigation of choice to exclude other causes of foramen of Monro obstruction and to assess the presence of potential membranes. In addition Cine-MRI CSF flowmetry is done to display CSF flow dynamics and velocity and to determine the site of CSF flow obstruction [73].

Neuroendoscopy is the treatment of choice as endosopic fenestration, foraminoplasty and septostomy can be done safely and effectively with this technique and spare the patient lifelong cumulative risk of shunt failure [74].

16. Choroid plexus tumors

16.1 Introduction

Choroid plexus tumors [CPTs] are rare intraventricular papillary tumors derived from choroid plexus epithelium which account for 0.3%–0.7% of all intracranial tumors, 2–6% of pediatric brain tumors, 10–20% of brain tumors in children less than 1 year and less than 1% of all adult intracranial tumors. The spectrum of choroid plexus tumors include a] WHO grade I choroid plexus papilloma b] WHO grade II atypical choroid plexus papilloma and c] WHO grade III choroid plexus carcinoma. Choroid plexus papillomas are more common than choroid plexus carcinomas in ratio of about 5: 1 [72, 75].

CPTs arise wherever choroid plexus tissue exists; the lateral ventricle being the most common site [50% of cases] followed by fourth ventricle [40%], third ventricle [5%] and multiple ventricles [5%] [75, 76].

16.2 Clinical features

CPTs most commonly present with signs of raised ICP and hydrocephalus [77–79]. Hydrocephalus can result due to overproduction of CSF, IVH, obstruction of CSF flow or impaired absorption. In addition to symptoms of raised intracranial pressure, patients may also present with seizures, focal neurological deficits, complications of chronic raised intracranial pressure like CSF rhinorrhea and visual disorientation, macrocephaly, and gait unsteadiness. Specific clinical symptoms can also be present according to the location of the tumor. Diencephalic seizures and bobble head doll syndrome- due to compression of the thalamus, signs of brainstem compression, cranial nerve palsies and cerebellar dysfunctions- due to posterior fossa CPTs, endocrine disturbances, precocious puberty, diabetes insipidus or diencephalic disorders- due to tumor in the third ventricle.

16.3 Neuroradiology

On Non contrast CT [NCCT], these tumors are seen as isodense to hyperdense intraventricular masses with intense contrast enhancement [72, 80].

MRI is the investigation of choice for these tumors. On MRI, CPTs appear as a large intraventricular lesion with irregular enhancing margins. They are isointense to slightly hyperintense on T1 weighted images, slightly hyperintense on T2 weighted images and show significant contrast enhancement.

16.4 Treatment

Maximum surgical resection followed by non-standardized use of adjuvant chemotherapy and radiotherapy is the treatment of choice for CPTs [75, 81].

17. Management of Foramen of Monro obstruction

17.1 Diagnosis of foramen of Monro obstruction

A detailed clinico-radiological examination is mandatory for diagnosis of foramen of Monro obstruction. Clinical manifestations and radiological features of

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various pathologies causing foramen of monro obstruction have already been discussed earlier in this chapter. Radiological features suggestive of foramen of monro obstruction include a] univentricular or biventricular hydrocephalus b] nonvisualization of the third ventricle c] obvious pathology seen in the third or lateral ventricle in the vicinity of the foramen.

17.2 Management

Nonoperative management: nonoperative management of foramen of Monro obstruction can be contemplated for very few indications like in patients with a small IVH causing hydrocephalus or in cases of idiopathic foramen of Monro obstruction with no signs of raised ICP.

17.3 Surgical management

Surgical management of the foramen of Monro obstruction depends on the pathology causing the obstruction. Approach can be done either by a] open craniotomy b] neuroendoscopy.

17.4 Open Craniotomy approaches

There are many surgical approaches for approaching tumors of the ventricular system. The open approach to foramen of Monro lesions depends on whether the lesion is predominant in the lateral or third ventricle. Microneurosurgical techniques used to reach the frontal horn of the lateral ventricles and foramen of Monro are a] frontal transcortical approach and b] anterior transcallosal approach [82]. Approach to foramen of monro lesions predominantly in third ventricle can be done by lamina terminalis approach either through the pterional or subfrontal corridors (**Figure 17**).



Figure 17. *Surgical approaches to the frontal horn of the lateral ventricle or third ventricle.*

18. Endoscopic approach

18.1 Endoscopic anatomy

Anatomical landmarks important to be recognized while performing neuroendoscopic approach to the lateral and third ventricle for performing septostomy and foraminoplasty include: the anterior caudate vein, thalamostriate vein, septal vein, choroid plexus and foramen of Monro (**Figure 18**).

18.2 Introduction

With recent advances in endoscopic techniques, neuroendoscopy has become the first line of management for intraventricular pathologies. Endoscopic approach to the foramen of Monro can be considered as minimally invasive version of the open transcortical approach with the added advantage of panoramic view which can be achieved by angling the scope or using scopes of different viewing angles. With recent advances in scope instrumentation, bimanual dissection of pathologies has also become possible. The definitive treatment of the lesion by endoscopic approach can always be supplemented with septostomy and monroplasty/foraminoplasty.

18.3 Septostomy

Endoscopic septostomy allows to bypass a unilateral foramen of Monro obstruction creating a CSF circulation between the obstructed ventricle and the opposite lateral ventricle that communicates with the third ventricle by the normal foramen of Monro [77]. This communication between both the lateral ventricles converts them into a single compartment thus allowing both the ventricles to be drained by one shunt in cases of bilateral foramen of Monro obstruction.

For performing a septostomy, a linear incision is generally taken 5-6cms lateral to the midline which is more lateral than the incision taken for endoscopic third ventriculostomy. The use of navigation also helps in deciding the site of the incision. A semicircular incision may be opted if we are planning to insert an Ommaya reservoir.

Authors recommendations: The site of septostomy on the septum pellucidum is generally posterosuperior to the foramen of Monro, posterior to the anterior septal vein, at the point where the septum appears to be avascular and thinned out. The



Figure 18. *Anatomy of the lateral ventricle.*



Figure 19. *A*] Septum pelucidum being probed in a avascular area B] post- perforation, dilatation of septostomy with balloon dilator C] opposite ventricular wall seen through septostomy.

probing of the septum with the blunt tip of the bipolar probe also gives the surgeon an idea of the thickness of septum pellucidum. If tumor biopsy or resection is planned simultaneously, septostomy should precede the biopsy or resection as bleeding while dealing with the tumor may obscure the anatomical orientation making the septostomy difficult (**Figure 19**).

Intraventricular hemorrhage has been documented as a complication in literature and the possible reason is injury to the contralateral septal vein during the septostomy being performed. However, no such experience has been encountered by the authors.

In a study by Oertel et al. septostomy was performed 5–10 mm posterior to the interventricular foramen, in the middle of the corpus callosum [CC] and the fornix [83]. In a study by Hamada et al. perforation of the septum was done between the anterior and posterior septal vein [78]. In a study by Roth et al. perforation was done in the anterior septal area, at the level of the interventricular foramen, midway between the corpus callosum and the fornix [79].

The largest study on the procedure of endoscopic septostomy was done by P.R. Aldana and their inference was that septostomy is the most adequate surgery for unilateral obstruction at the interventricular foramen level [84].

19. Endoscopic Foraminoplasty

Endoscopic foraminoplasty at the interventricular foramen level is not very commonly reported in the literature. It was reported for the first time by Oi et al. [85]. This procedure establishes back the connection between the lateral and the third ventricle. It obviates the need of a shunt and avoids its related complications. For performing foraminoplasty, the incision and entry of the scope is similar to that of septostomy.

On entry to the ventricle, the foramen of Monro is recognized by choroid plexus and the thalamostriate and septal veins.

Endoscopic foraminoplasty can be performed by:

- a. excision of the offending lesion
- b. clearance of the exudates, adhesions or blood products

- c. perforation of septations or membranes
- d. enlargement with balloon catheter or dilating forceps



Figure 20.

for aminoplasty can be done by A] excision of lesion B] removal of blood products C] removal of exudates D] removal of adhesions E] dilatation with dilator forceps F] dilatation with balloon catheter.



Table 4.Algorithm for surgical management of foramen of Monro obstruction.

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As it always carries a risk of recurrent obstruction because of the scarring, foraminoplasty should be supplemented with a septostomy. The procedure of dilatation carries the risk of injury to the septal vein or thalamostriate vein and there can also be injury to the fornix leading to memory problems. However, in our experience, we have never encountered vascular injury in any of our cases. In 1 case, injury to the fornix was seen. However, postoperatively, the patient did not experience any memory problems (**Figure 20**) (**Table 4**).

20. Conclusion

Lesions causing foramen of Monro obstruction resulting in hydrocephalus are a fairly uncommon entity encountered in neurosurgical practice with craniopharyngioma and colloid cysts being the most common pathology in children and adults respectively.

Treatment consists of open craniotomy for solid tumors and endoscopic approaches (transnasal transsphenoidal and cranial) for cystic lesions. Endoscopic approach is particularly helpful in decreasing the convalescence period and postoperative complications and thus, should be offered as a first line of treatment whenever suited.

Septostomy should be a part of standard treatment in all the patients having foramen of Monro obstruction so as to obviate the need of added shunt procedures.

Although small solid lesions less than 2 cms in size can be addressed by endoscopic approach, the learning curve required for endoscopic approach to deal with solid lesions is very steep. Thus, correct patient selection is of utmost importance for optimal patient outcome.

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