Brain tumor

&

Raised intracranial pressure

By

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Brain tumors

Brain tumors are responsible for approximately 2% of all cancer deaths. Central nervous system tumours comprise the most common group of solid tumours in young patients, accounting for 20% of all paediatric neoplasms. The overall incidence of brain tumours is 8–10 per 100 000 population per year.

Glioma

Neuroectodermal tumors arise from cells derived from neuroectodermal origin. Gliomas comprise the majority of cerebral tumours and arise from the neuroglia cells. There are four distinct types of glial cells: astrocytes, oligo-dendroglia, ependymal cells and neuroglial precursors. Each of these gives rise to tumours with different biological and anatomical charac-teristics. The neuroepithelial origin of microglia is in question.

Classification of brain Tumour

1.NEUROEPITHELIAL TUMOUR
Gliomas:
Astrocytoma
Oligodendrocytoma
Ependymoma
Choroid plexus Tumour

Pineal Tumors
Neuronal Tumour :ganglioglioma,gangliocytoma,neuroblastoma
Medulloblastoma

2.NERVE SHEATH TUMOUR:acoustic neuroma
3.MENINGEAL TUMOUR:meningioma
4.PITUITARY TUMOUR
5.GERM CELL TUMOURS:germinoma,teratoma
6.LYMPHOMAS
7.TUMOUR LIKE MALFORMATION:
craniopharyngioma,epidermoid,dermoid,colloid cyst
8.METASTATIC TUMORS
9.LOCAL EXTENSIONS FROM REGIONAL TUMOUR: glomus jugular,carcinomas of ethmoid

Neuroepithelial tumours
Gliomas
  Astrocytoma (including glioblastoma)
  Oligodendrocytoma
  Ependymoma
  Choroid plexus tumour
Pineal tumours
Neuronal tumours
  Ganglioglioma
  Gangliocytoma
  Neuroblastoma
Medulloblastoma

Nerve sheath tumour—acoustic neuroma

Meningeal tumours
Meningioma

Pituitary tumours

Germ cell tumours
Germinoma
Teratoma

Lymphomas

Tumour-like malformations
Craniopharyngioma
Epidermoid tumour
Dermoid tumour
Colloid cyst

Metastatic tumours

Local extensions from regional tumours
e.g. glomus jugular (i.e. jugulare), carcinoma of ethmoid

Ringertz	Grade I (Well differentiated)	Grade II (Anaplastic astrocytoma)	Grade III (Glioblastoma multiforme)	
Kernohan	Grade I	Grade II	Grades III and IV	
WHO	Grade I Juvenile pilocytic astrocytoma	Grade II Astrocytoma variants • Fibrillary • Protoplasmic • Gemistocytic	Grade III Anaplastic astrocytoma	Grade IV Glioblastoma variants • Giant cell • Gliosarcoma
St Anne-Mayo	Grade I Score: 0	Grade II Score: 1	Grade III Score: 2	Grade IV Score: 3 or 4

Relationship of the Kernohan system, the 'three-tiered' classification system, the WHO system and the St Anne/Mayo (Dumas-Deport) grading system for astrocytomas.

Pathology
Macroscopic changes

An astrocytoma may arise in any part of the brain, although it usually occurs in the cerebrum in adults and the cerebellum in children.

A low-grade tumour in the cerebral hemi- spheres invades diffusely into the brain. The tu- mour does not have a capsule and there is no distinct tumour margin. The low-grade gliomas are usually relatively avascular with a firm fibrous or rubbery consistency

the macroscopic appearance of a high- grade tumour, the glioblastoma multiforme, is characterized by a highly vascular tumour margin with necrosis in the centre of the tumour

Microscopical

The low-grade astrocytoma is characterized by an increased cellularity, composed entirely of astrocytes (Fig. 6.2). Intermediate-grade tumours show nuclear pleomorphism, mitotic figures are frequent, and there is increased vascularity,

The major histological features of glioblastoma multiforme are endothelial proliferation and necrosis. The anaplastic astrocytoma is char-acterized by nuclear pleomorphism and mitoses, which are absent in the astrocytoma.

Clinical presentation

The presenting features can be classified under: • raised intracranial pressure
• focal neurological signs
• epilepsy.

The duration of the symptoms and the progression and evolution of the clinical presentation will depend on the grade of the tumour —that is, its rate of growth.

patient presenting with a low-grade astrocytoma (Grade I or II) may have a history of seizures extending over many years

Patients with the higher-grade tumours present with a shorter history and glioblastoma multiforme is characterized by a short illness of weeks or a few months.

Raised intracranial pressure

Raised intracranial pressure is due to the tumour mass, surrounding cerebral edema and hydrocephalus due to blockage of the CSF pathways

The major symp- toms are headache, nausea and vomiting, and drowsiness.

Headache is the most common symptom in patients with cerebral astrocytoma and occurs in nearly three-quarters of patients; vomiting occurs in about one-third. The headaches are usually gradually progressive and although frequently worse on the side of the tumors, they may be bitemporal and diffuse. Characteristically, the headache is worse on waking and improves during the day. Nausea and vomiting occur as the intracranial pressure increases, and the patient frequently indicates that vomiting may temporarily relieve the severe headache. Drowsiness, that is, a deterioration of conscious state, is the most important symptom and sign of raised intracranial pressure. The extent of impairment of conscious state will be related to the severity of raised intracranial pressure. An alert patient with severely raised intracranial pressure may rapidly deteriorate and become deeply unconscious when there is only a very small further rise in the pressure within the cranial cavity.

Focal neurological deficit

Patients presenting with tumours involving the frontal lobes frequently have pseudopsychiatric problems, personality change and mood disturbance. These changes are particularly char- activistic of the 'butterfly glioma', so called because it involves both frontal lobes by spreading across the corpus callosum, giving it a characteristic macroscopic

Limb paresis

Field defects associated with Tumors of the temporal, occipital and parietal lobes are common, but may be evident only on careful testing

Dysphasia

Epileptic seizures

Seizures are the most frequent initial symptom in patients with cerebral astrocytoma and occur in 50–75% of all patients. Tumours adjacent to the cortex are more likely to be associated with epilepsy than those deep to the cortex and tumours involving the occipital lobe are less likely to cause epilepsy than those which are more anteriorly placed.

Investigations

Computerized tomography

CT scan or MRI of the brain are the essential radiological investigations an accurate diagnosis can be made in nearly all tumours. Low-grade gliomas show decreased density on the CT scan; this does not enhance with contrast and there is little or no surrounding oedema. Calcification may be present. High-grade gliomas are usually large and enhance vividly following intravenous injection of contrast material . The enhancement is often patchy and non- uniform and frequently occurs in a broad, irregular rim around a central area of lower density. Although tumour cysts may occur in the high- grade tumours, the central area of low density surrounded by the contrast enhancement is usually due to tumour necrosis. High-grade tumours are surrounded by marked cerebral oedema and there is frequently considerable distortion of the lateral ventricles. Compression of the lateral ven- tricle in one hemisphere, with pressure extending across the midline, may result in an obstructive hydrocephalus involving the opposite lateral ventricle.

MRI

When used with gadolinium contrast enhancement, MRI improves the visualization and anatomical localization of the tumours (Figs 6.8 and 6.9). MRI has the advantage of being more sensitive than CT scan, enabling the detection of small tumours and particularly low-grade gliomas that might be missed by CT scan. MRI provides better anatomical detail and is more useful in visualizing skull base, posterior fossa and brainstem tumours.

Cerebral angiography

This was the standard study in most patients with astrocytomas prior to the introduction of CT. It provides helpful information on the vascular supply of the tumours but is now only rarely indicated.

Plain X-rays

Plain X-rays of the skull do not need to be performed as a routine. The most common abnormality is erosion of the sella turcica due to long-standing raised intracranial pressure. Radiologically visible calcification is present in about 8% of patients with astrocyte-derived gliomas.

Management

Following the presumptive diagnosis of a glioma the management involves:

• surgery

• radiotherapy

• other adjuvant treatments.

Surgery

Surgery is performed with three principal aims. • To make a definite diagnosis.

• Tumour reduction to alleviate the symptoms of raised intracranial pressure.

• Reduction of tumour mass as a precursor to adjuvant treatments.

The patient is started on glucocorticoid steroid therapy (e.g. dexamethasone) when presenting with clinical features of raised intracranial pressure with the aim of decreasing the cerebral oedema prior to surgery.

Other adjuvant therapies

Chemotherapy

Radiotherapy

Hyperthermia

Immunotherapy

Photodynamic therapy

Gene therapy

Oligodendroglioma

Oligodendrogliomas are responsible for approximately 5% of all gliomas and occur throughout the adult age group with a maximal incidence in the 5th decade. The tumour is rare in children.

Pathology

Oligodendrogliomas have the same spectrum of histological appearance as astrocytomas, rang- ing from very slow growing, benign tumours to a more rapidly growing, malignant variety with abundant mitotic figures, endothelial prolifera- tion and foci of necrosis. Calcium deposits are found by histological examination in up to 90% of oligodendrogliomas

Clinical presentation

The presenting features are essentially the same as for the astrocyte group but, as these tumours are more likely to be slow growing, epilepsy is common, occurring in 80% of patients and seen as an initial symptom in 50%. The features of raised intracranial pressure and focal neurologi- cal deficits are each present in approximately one-third of patients.

Radiological investigation

CT scanning and MRI are the fundamental inves- tigations. They will confirm the diagnosis of an intracranial tumour and in many cases the diag- nosis of oligodendroglioma will be highly probable. Calcification will be present in 90% of cases and over half show contrast enhancement

Treatment and results

Treatment involves:

- *surgicalresection*
- *radiotherapy*
- *otheradjuvanttreatments.*

The standard treatment for oligodendroglioma has been an aggressive resection of the tumour followed by radiation therapy, although radio- therapy would now not be given to low-grade tu- mours, and utilized only for the intermediate- or high-grade oligodendroglial tumours. Oligoden- drogliomas have been shown to be more sensi- tive to chemotherapy than the astrocytoma tumours,

Ependymoma

Ependymomas are glial neoplasms arising from the ependyma and constitute approximately 5% of all gliomas. Approximately two-thirds of ependymomas occur in the infratentorial com- partment and most of these present in children, adolescents and young adults. The supraten- torial ependymomas occur mostly in adults.

Pathology

The tumour arises from the ependyma of the ven-

tricle and, although predominantly intraventric- ular, the tumour often invades into the adjacent cerebellum, brainstem or cerebral hemisphere

Clinical presentation

Posterior fossa ependymomas

Patients present with features of raised intracranial pressure due to hydrocephalus as a result of obstruction of the 4th ventricle, ataxia due to cerebellar involve- ment, and occasionally features of brainstem pressure or infiltration.

Supratentorial tumours

Virtually all patients with supratentorial ependy- momas present with features of raised intracranial pressure, often due to hydrocephalus as a result of obstruction of the CSF pathways. Ataxia is common and focal neurological deficits may occur due to involvement of the underlying cere- bral hemisphere.

Radiological investigation

The CT scan and MRI will show a tumour that arises in the ventricle and enhances after admin- istration of intravenous contrast. Calcification is common in tumours arising from the lateral ventricles. .. There is frequently associated hydrocephalus

Treatment

The treatment of ependymomas is initially surgical, with an attempt to perform a radical macroscopic resection of the tumour

Postoperative radiation therapy is advisable and, as these tumours may spread through the CSF.pathways, sometimes whole neuraxis radiation is recommended.

The prognosis is related to the degree of anaplasia of the tumour and for intratentorial tu- mours varies from 20% to 50% 5-year survival. The prognosis for the supratentorial tumours is better, particularly in adults.

Pineal Tumours

·germinoma
· teratoma
· pineocytoma · pineoblastoma · miscellaneous:·
glioma ·cyst

Germinoma is the most common pineal region tumour and is similar in histological appearance to germinoma of the gonads and mediastinum; it occurs predominantly in males.

Clinical presentation

Patients with pineal tumours present with:
· raised intracranial pressure
· neurological signs dueto focal compression ·endocrine disturbance.

Neurological signs

ataxia and distortion of the quadrigeminal plate, produces limitation of upgaze, convergence paresis with impairment of reaction of pupils to light and accommodation (Parinaud's syndrome), and may result in convergence-retraction nystagmus on upgaze (Koerber–Salius–Elschnig syndrome).

Endocrine disturbance. ***are uncommon but include precocious puberty in 10% of patients, almost invariably male, and diabetes insipidus in 10%. The endocrine effects can either be due to direct tumour involvement of the hypothalamus or result from the secondary effects of hydrocephalus.***

Radiological investigations

CT scan and MRI will show a pineal region tumour and will often suggest the correct pathol- ogical diagnosis

Management

This consists of surgery and radiotherapy.
A ventriculoperitoneal shunt or drainage of CSF by a 3rd ventriculostomy may be required if

the hydrocephalus is severe.

Metastatic tumours

Metastatic tumours are responsible for approxi- mately 15% of brain tumours in clinical series but up to 30% of brain tumours reported by patholo- gists

carcinoma of the lung

carcinoma of the breast

metastatic melanoma

carcinoma of the kidney gastrointestinal carcinoma

The presenting features are similar to those described for other intracranial tumours:

- ***raisedintracranialpressure***
- ***focalneurologicalsigns***
- ***epilepticseizures.***

Radiological investigations

CT scan or MRI will diagnose the metastatic tumour and will show whether the deposits are solitary or multiple

Treatment

***Steroid** medication (e.g. dexamethasone) will control cerebral oedema and should be commenced immediately if there is raised intracra- nial pressure.*

***Surgery** to remove the metastasis is indicated if: • there is a solitary metastasis in a surgically accessible position • there is no systemic spread.*

***Radiotherapy**, together with steroid medication to control cerebral oedema, is used to treat patients with multiple cerebral metastases and may be advisable following the excision of a single metastasis*

Leptomeningeal metastases

Meningeal carcinomatosis is widespread, multi- focal seeding of the leptomeninges by systemic cancer.

Chordomas

Chordomas are rare tumours arising from noto- chord cell nests. They may arise throughout the craniospinal axis but occur predominantly at the ends of the axial skeleton in:

- ***thebasioccipitalregion***
- ***thesacrococcygealregion.***

Clinical presentation

The majority of intracranial chordomas arise be- tween 20 and 60 years of age. The clinical features result from the widespread tumour extension and include:

- ***raised intracranial pressure, causing headaches and vomiting***
- ***multiple cranial nerve palsies, often unilateral • nasopharyngeal obstruction.***

Posterior fossa tumours

Sixty per cent of paediatric brain tumours occur in the posterior fossa. The relative incidence of the tumours is:

1 cerebellarastrocytoma30%

2 medulloblastoma (infratentorial neuroectodermal tumour) 30%

3 ependymoma 20%

4 brainstem glioma 10%

5 miscellaneous 10%:
(a) choroid plexus papilloma (b)haemangioblastoma
(c) epidermoid, dermoid
(d) chordoma.

Meningioma

The tumour arises from the arachnoid layer of the meninges, principally the arachnoid villi and granulations

Meningiomas are the most common of the benign brain tumours and constitute about 15% of all in- tracranial tumours, being about one-third of the number of gliomas. Although they may occur at any age, they reach their peak incidence in mid- dle age, are very uncommon in children and occur more frequently in women than men.

The major histological types are:

Syncytial or meningotheliomatous

The transitional type

The fibroblastic type

Angiomatous meningiomas

Malignant meningiomas

Clinical presentation

Meningiomas present with features of: • raisedintracranialpressure • focalneurologicalsigns • epilepsy.

Radiological investigations

The CT scan appearance shows a tumour of slightly increased density prior to contrast; it enhances vividly and uniformly following intravenous contrast. Hyperostosis of the cranial vault may be a focal process at the site of the tumour attachment or, as seen with en plaque meningioma, a more diffuse sclerosis. These bone changes may also be seen on plain skull X-ray.

Magnetic resonance imaging will demonstrate meningiomas following the intravenous injection of gadolinium contrast

Preoperative management

Meningiomas are frequently surrounded by severe cerebral oedema and patients should be treated with high-dose steroids (dexamethasone) prior to surgery if possible. Preoperative embolization of the tumour vasculature may be considered advisable in some anterior basal and sphenoidal wing tumours where the major vascular supply is not readily accessible in the early stages of the operation.

Treatment

The treatment of meningiomas is total surgical excision, including obliteration of the dural at- tachment. Although this objective is usually pos- sible there are some situations where complete excision is not possible because of the position of the tumour. Tumours arising from the clivus, in front of the brainstem or those situated within the cavernous sinus, are notoriously difficult to excise without causing serious morbidity.

***Radiation** therapy may be used to treat residual tumours following subtotal resection, in order to reduce the risk of recurrent growth.*

***Stereotactic radiotherapy** has been used to treat small meningiomas (less than 3 cm in diam- eter), particularly if the tumours are located in portions not easily amenable to surgery, or in the elderly or medically infirm patient.*

Acoustic neuroma

Acoustic schwannomas arise from the 8th cranial nerve and account for 8% of intracranial tumours

CPA in decreasing frequency, are:

- ***meningioma***
- ***metastatic tumour***
- ***exophytic brainstem glioma*** - ***epidermoid tumour.***

Clinical manifestations of pituitary tumours.

'Mass' effects

Headaches (especially acromegaly) Superior extension

Chiasmal syndrome (impaired visual acuity and fields)

Hypothalamic syndrome (disturbance in thirst, appetite, satiety, sleep and temperature regulation; diabetes insipidus —uncommon; inappropriate ADH syndrome —uncommon)
Obstructive hydrocephalus Lateral extension

Cranial 3rd, 4th, 6th, diplopia Cranial 5th, facial pain Temporal lobe dysfunction
Inferior extension Nasopharyngeal mass CSF rhinorrhoea

'Endocrine' effects
Hyperpituitarism
GH —gigantism/acromegaly
PRL —hyperprolactinaemic syndrome ACTH —Cushing's disease
TSH —thyrotoxicosis

Hypopituitarism
GH —child: shortness of stature,

hypoglycaemia
PRL —adult female: failure of postpartum

lactation
ACTH —hypocortisolism (Addison's) TSH —hypothyroidism
LH/FSH —hypogonadism

Acute deterioration
Pituitary apoplexy

Treatment of pituitary Tumour

1 Operative procedures:

(a) trans-sphenoidal excision (b) transcranial excision.

2 Radiotherapy.

3 Medical treatment with antisecretory drugs.

Craniopharyngioma

This tumour may occur at any age, although nearly half occur in the first 20 years of life. They are thought to arise from the epithelial remnants of Rathke's pouch.

The tumours occur in the region of the pitu- itary fossa and extend through the suprasellar cisterns to the hypothalamus. The majority are cystic, and the fluid is often yellow and sparkling with cholesterol crystals.

The cyst may be larger than the solid component, which is often pale and crumbly, consisting of epithelial debris.

adamantinous type resembles adamantino- ma of the jaw and is encountered in virtually all children. The papillary type, so-called adult craniopharyngioma, occurs in about one-third of adults and is rare in children.

Raised intracranial pressure

It is a most important neurological condition, requiring prompt diagnosis and often needing urgent treatment.

The normal supine intracranial pressure is 10–15 mmHg, measured at a position equal to the level of the foramen of Monro

Raised intracranial pressure may be due to:

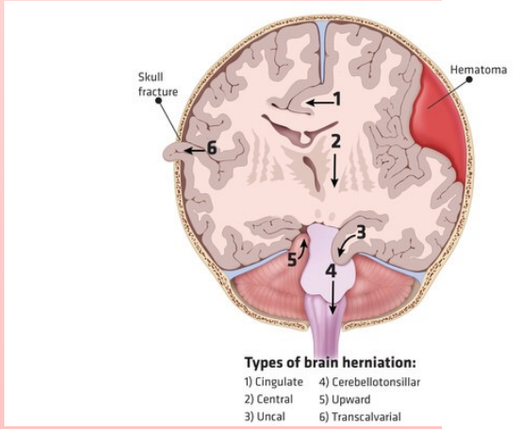
• increased volume of normal intracranial constituents
• space occupying lesion.

The increase in volume of normal intracranial contents may be due to:
•••• brain
• cerebral edema
• benign intracranial hypertension

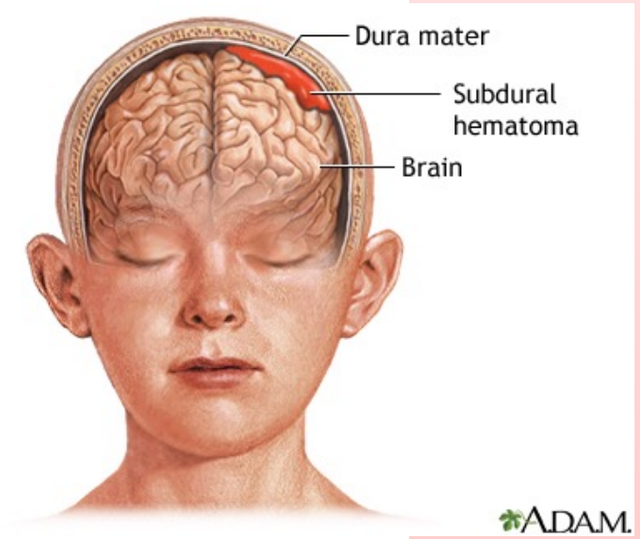
••• CSF
• hydrocephalus
••• blood volume.
• vasodilatation

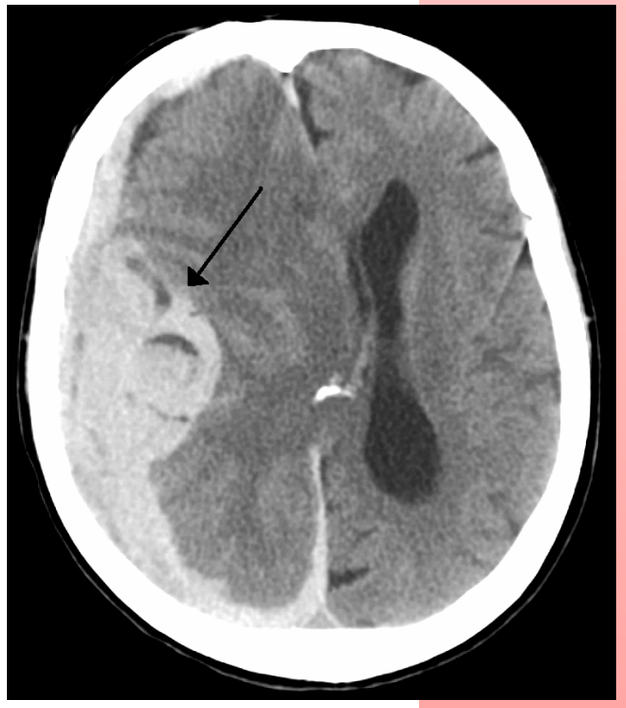
Table 1 Causes of raised intracranial pressure

- Increased brain volume
  - Intracranial space occupying lesions
    - Brain tumors
    - Brain abscess
    - Intracranial hematoma
    - Intracranial vascular malformation
  - Cerebral edema
    - Encephalitis (viral, inflammatory)
    - Meningitis
    - Hypoxic ischemic encephalopathy
    - Traumatic brain injury
    - Hepatic encephalopathy
    - Reye's syndrome
    - Stroke
    - Reye's syndrome
- Increase in CSF volume
  - Hydrocephalous
  - Choroids plexus palpilloma
- Increased blood volume
  - Vascular malformations
  - Cerebral venous thrombosis
  - Meningitis, encephalitis



Types of brain herniation:
1) Cingulate 4) Cerebellotonsillar
2) Central 5) Upward
3) Uncal 6) Transcalvarial





Cerebral blood flow

Between physiological ranges in blood pressure, the brain is able to maintain a constant cerebral blood flow. This is achieved by a process called autoregulation whereby the brain adjusts the in-tracranial vascular resistance by altering vessel diameter and tone

CPP=MAP-ICP

Thus in order to maintain cerebral perfusion in the presence of raised ICP, the systemic blood pressure needs to be elevated.

Intracranial Causes	Extra-Cranial Causes
Intracranial masses	**Conditions leading to generalized brain swelling**
• Infarction (stroke) with edema	• Hypoxia (e.g. acute mountain sickness)
• Traumatic injuries with edema	• Hypertensive encephalopathy
• Hemorrhages (spontaneous or traumatic)	• Acute liver failure
• Brain tumors	• End-stage kidney failure
• Brain abscesses	• Hypercarbia (e.g. chronic pulmonary disease)
CSF accumulation	**Increased intracranial venous pressure**
• Increased CSF production (e.g. tumors of the choroid plexus)	• Cavernous sinus thrombosis
• Obstruction to CSF flow (non-communicating hydrocephalus)	• Obstruction of jugular veins • Superior vena cava syndrome
• Impaired CSF resorption (communicating hydrocephalus)	• Right heart failure

Clinical symptoms and signs of raised intracranial pressure

The common causes of raised intracranial pressure are:

- ***space-occupying lesion—cerebral tumour (and edema), abscess, intracranial hematoma***
- ***hydrocephalus***
- ***benign intracranial hypertension.***

The clinical features will be determined in large part by the underlying cause of the raised pressure. However, some of the clinical symptoms and signs will be the same, no matter what the cause of the raised pressure. The major features are:

- ***headache***
- ***nausea and vomiting***
- ***drowsiness***
- ***papilloedema.***

Table 3.1 Transtentorial herniation.

Compression of 3rd cranial nerve—causing initial dilatation of the ipsilateral pupil
Compression of the midbrain Hemiparesis, usually contralateral Occasional compression of opposite crus cerebri causes ipsilateral hemiparesis Hypertension, bradycardia—Cushing response Respiratory failure
Compression of posterior cerebral artery

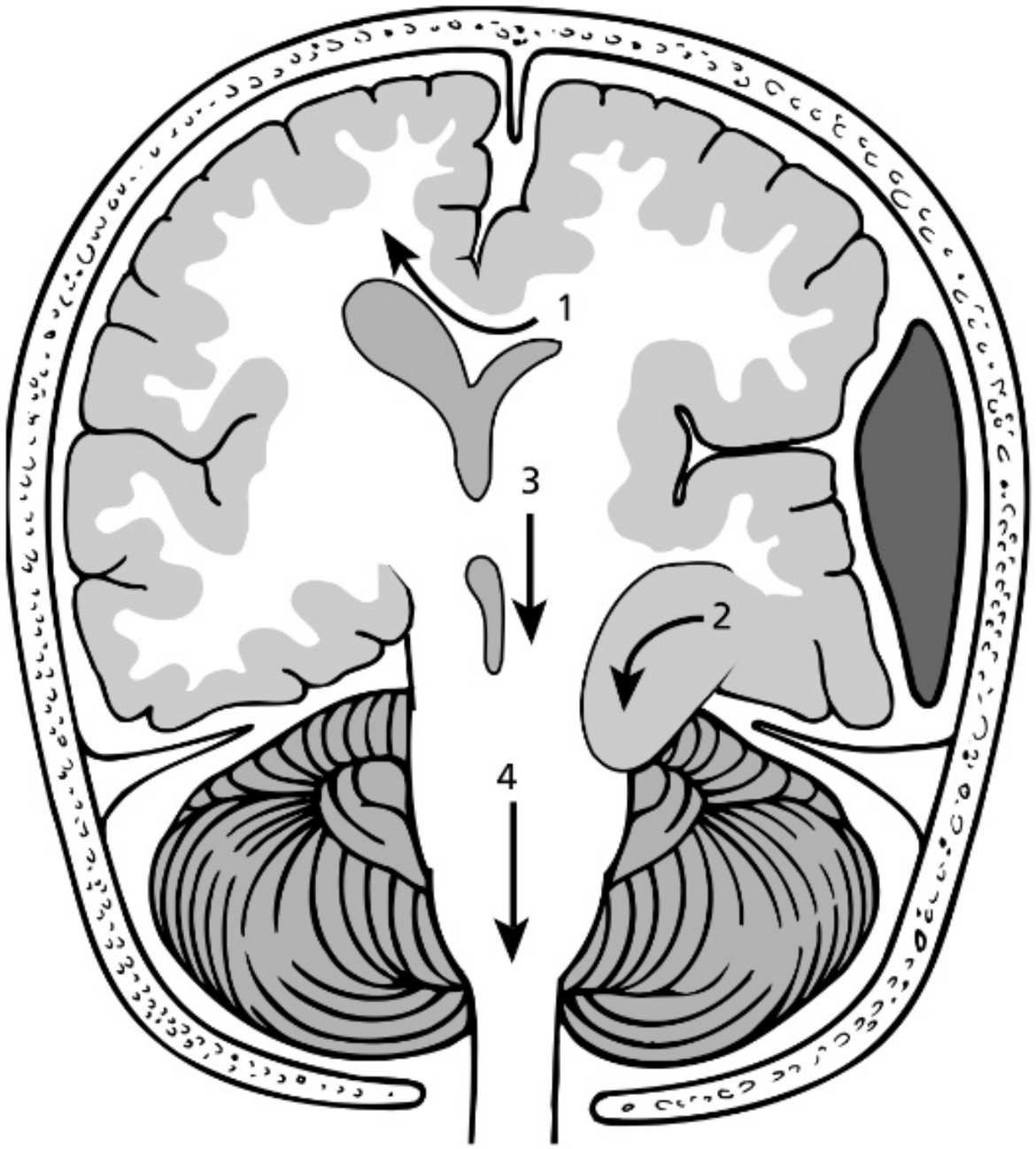


Fig. 3.3 Brain herniations. A lateral supratentorial mass will cause displacement of the lateral ventricles with: (1) subfalcine herniation of the cingulate gyrus below the falx cerebri; (2) herniation of the uncus into the tentorial hiatus; (3) caudal displacement of the brainstem. Raised pressure within the posterior fossa may cause herniation of the cerebellar tonsils into the foramen magnum (4). (Adapted from Jennett &

Headache. ***The headache associated with in- creased intracranial pressure is usually worse on waking in the morning and is relieved by vomiting. Intracranial pressure increases during sleep, probably from vascular dilatation due to carbon dioxide retention. The cause of the headache in raised intracranial pressure is probably traction on the pain-sensitive blood vessels and compres- sion of the pain-sensitive dura at the base of the cranium.***

Nausea and vomiting. ***The nausea and vomiting is usually worse in the morning.***

Papilloedema. ***The definitive sign of raised intracranial pressure, papilloedema is due to transmission of the raised pressure along the subarachnoid sheath of the optic nerve***

Long-standing papilloedema ***from prolonged raised intracranial pressure will subsequently develop into secondary optic atrophy.***

Cushing reflex:

Hypertension/bradycardia/irregular respiration

Sixth nerve palsy, ***causing diplopia, may occur in raised intracranial pressure due to stretching of the 6th nerve by caudal displacement of the brainstem.***

In an infant, raised intracranial pressure will cause a tense, bulging fontanelle.

Measurement of intracranial pressure

The most common indications are:

- ***Head injury***
- ***Following major intracranial surgery, when measurement of the intracranial pressure may help in the management of patients***

- ***In the assessment of dementia and benign intracranial hypertension***

The intracranial pressure may be recorded from the ventricle, brain substance, subdural or extradural space. ***The intracranial catheters are attached by a transducer to a continuous recorder. There are now numerous monitoring devices with various degrees of technical sophistication.***

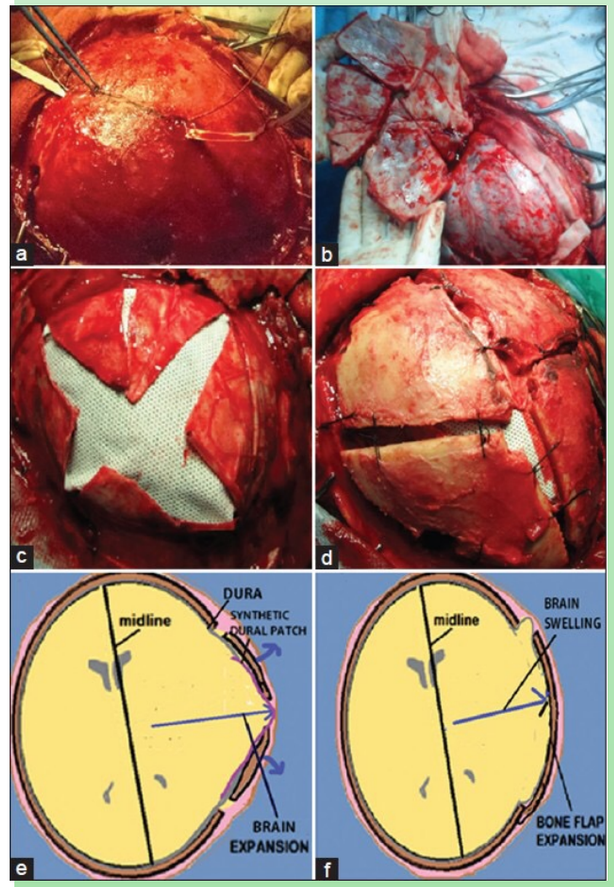
Management of raised intracranial pressure

The treatment of raised intracranial pressure will depend on the underlying cause,This may involve resection of a space-occupying lesion, or in the case of hydrocephalus, a CSF shunt

In an emergency situation, when the patient has become comatose and has failing respiration, it is essential that the patient's ventilatory state is urgently maintained and this will necessitate the passage of an endotracheal tube and ventilatory support. While the patient is being transferred for definitive treatment of the raised pressure it may be possible to temporarily lower the intracranial pressure by hyperventilation which will reduce arterial CO2 and diminish vasodilatation, and by the administration of a diuretic such as mannitol or frusemide (furosemide)

Table 3 Summary of measures to reduce intracranial pressure

1	Assessment and management of ABC's (airway, breathing, circulation)
2	Early intubation if; GCS <8, Evidence of herniation, Apnea, Inability to maintain airway
3	Mild head elevation of 15–30° (Ensure that the child is euvolemic)
4	Hyperventilation: Target $PaCO_2$: 30–35 mm Hg (suited for acute, sharp increases in ICP or signs of impending herniation)
5	Mannitol: Initial bolus: 0.25–1 g/kg, then 0.25–0.5 g/kg, q 2–6 h as per requirement, up to 48 h
6	Hypertonic Saline: Preferable in presence of Hypotension, Hypovolemia, Serum osmolality >320 mOsm/kg, Renal failure, Dose: 0.1–1 ml/kg/hr infusion, Target Na^+−145–155 meq/L.
7	Steroids: Intracranial tumors with perilesional edema, neurocysticerocosis with high lesion load, ADEM, pyomeningitis, TBM, Abscess Acetazolamide: Hydrocephalous, benign intracranial, high altitude illness
8	Adequate sedation and analgesia
9	Prevention and treatment of seizures: use Lorazepam or midazolam followed by phenytoin as initial choice.
10	Avoid noxious stimuli: use lignocaine prior to ET suctioning [nebulized (4% lidocaine mixed in 0.9% saline) or intravenous (1–2 mg/kg as 1% solution) given 90 sec prior to suctioning]
11	Control fever: antipyretics, cooling measures
12	Maintenance IV Fluids: Only isotonic or hypertonic fluids (Ringer lactate, 0.9% Saline, 5% D in 0.9% NS), No Hypotonic fluids
13	Maintain blood sugar: 80–120 mg/dL
14	Refractory raised ICP: • Heavy sedation and paralysis • Barbiturate coma • Hypothermia • Decompressive craniectomy




Thank You