Clinical presentation and diagnosis of brain tumors

INTRODUCTION — Brain tumors are a diverse group of neoplasms arising from different cells within the central nervous system (CNS) or from systemic tumors that have metastasized to the CNS. Brain tumors include a number of histologic types with markedly different tumor growth rates (table 1 and table 2). (See "Classification of gliomas" and "Overview of the clinical manifestations, diagnosis, and management of patients with brain metastases").

Brain tumors can produce symptoms and signs by local brain invasion, compression of adjacent structures, and increased intracranial pressure (ICP). In addition to the histology of the tumor, the clinical manifestations are determined by the function of the involved areas of the brain. The proper evaluation of the patient with a suspected brain tumor requires a detailed history, comprehensive neurologic examination, and appropriate diagnostic neuroimaging studies [1].

The clinical manifestations and diagnosis of primary brain tumors will be reviewed here. Aspects of the various primary brain tumors are discussed in the specific topics, and the presentation and approach to patients with brain metastases is presented separately. (See "Overview of the clinical manifestations, diagnosis, and management of patients with brain metastases").

CLINICAL MANIFESTATIONS — Patients with primary or metastatic brain tumors may present with either generalized or focal signs and/or symptoms (table 3).

Generalized

Headaches — Headache is a common manifestation of brain tumors and is the worst symptom in about one-half of patients [2]. The headaches are usually dull and constant, but occasionally throbbing. Severe headaches are infrequent, unless obstructive hydrocephalus or meningeal irritation is present. The "classic" early morning brain tumor headache appears to be uncommon. (See "Evaluation of headache in adults").

Features suggestive of a brain tumor in a patient complaining of headaches include nausea and vomiting (present in about 40 percent), a change in prior headache pattern, and an abnormal neurologic examination [2]. In addition, many patients with a brain tumor report worsening of headache after a change in body position, such as bending over, or with maneuvers that raise intrathoracic pressure, such as coughing, sneezing, or the Valsalva maneuver [2]. These activities lead to a period of raised ICP, which uncommonly can cause a loss of consciousness (see 'Syncope' below).

The headache can be localizing. In the above series, the typical headache was bifrontal, but worse on the same side as the tumor [2]. Alternatively, a generalized headache may result from increased intracranial pressure (ICP), which can arise either from a large mass or from restriction of cerebrospinal fluid outflow causing hydrocephalus. Increased ICP may lead to the classic triad of headache, nausea, and papilledema. (See "Evaluation and management of elevated intracranial pressure in adults").
Tumor-related headaches tend to be worse at night and may awaken the patient. This nocturnal pattern is thought to be due in part to transient increases in PCO2, a potent vasodilator, during sleep. Other possible physiologic explanations include recumbency and decreased cerebral venous return.

The manifestations of headache in brain tumor patients were illustrated by a retrospective review of 111 patients with brain tumors, in which headaches were present in 48 percent of cases with either primary or metastatic disease [2]. The headaches were tension-type in 77 percent, migraine-type in 9 percent, and other types in 14 percent.

Brain tumor headaches are discussed in more detail elsewhere. (See “Brain tumor headache”.)

Seizures — Seizures are among the most common symptoms of gliomas and cerebral metastases. Some patients who are seizure-free at diagnosis subsequently develop seizures [3,4], but routine prophylaxis with anticonvulsant medications is not recommended. (See “Seizures in patients with primary and metastatic brain tumors”.)

The incidence of seizures is higher with primary tumors than with metastatic lesions, and among patients with primary tumors, seizures are less common with high-grade as opposed to low-grade gliomas [5,6]. This was illustrated in a review of 1028 patients with primary brain tumors: the prevalence of seizures was 49, 69, and 85 percent among patients with glioblastoma (GBM), anaplastic glioma, and low-grade glioma, respectively [6].

Seizures may be the presenting symptom or develop subsequently. In two large series of patients with GBM, seizures were the initial manifestation in 18 percent and were present at the time of diagnosis (for an average of one year) in 29 percent (table 4) [7,8]. The frequency and onset of seizures in patients with brain metastases was illustrated in a series of 195 patients, in which seizures were present at diagnosis in 9 percent and subsequently developed in another 10 percent [4].

Although seizures can be either generalized or focal, any seizure focus can cause a generalized seizure. In patients who have focal seizures, the clinical presentation is dependent upon the tumor location. As an example, frontal lobe tumors may cause focal tonic-clonic movements involving one extremity, while seizures originating within the occipital lobe may cause visual disturbances. Temporal lobe seizures are the most difficult to diagnose and localize. In this setting, abrupt sudden behavioral changes may occur with or without typical preseizure auras, such as abnormal smell, taste, or gastrointestinal symptoms.

Tumor-related seizures are typically repetitive and are stereotyped in a given patient. The ictal event may be preceded by an aura, which is itself a simple partial seizure, and followed by postictal sequela. The postictal phase can be manifested as a period of fatigue and an urge to sleep. For patients with focal seizures, a postictal paresis (also known as a Todd's paralysis) may be present. (See “Overview of the management of epilepsy in adults”.)

Both primary and metastatic brain tumors can cause status epilepticus, which may occur at the time of tumor diagnosis or subsequently [9]. (See “Status epilepticus in adults”.)

Patients who present with seizures usually have smaller primary brain tumors or fewer metastatic lesions in the brain, compared to those who present with other symptoms. This is probably because the diagnosis of a seizure is an indication for neuroimaging, which leads to an earlier diagnosis. (See “Evaluation of the first seizure in adults”.)

A seizure should not be confused with a syncopal event that may be caused by an abrupt increase in ICP. This distinction is important since treatment is different in the two conditions. Patients with seizures are at risk of subsequent seizures [3,10], and the treatment is anticonvulsants. In contrast, patients with increased ICP require urgent corticosteroids, neurosurgical intervention, or both. (See “Syncope” below and “Overview of the management of epilepsy in adults” and “Management of vasogenic edema in patients with primary and metastatic brain tumors”.)
**Nausea and vomiting** — Brain tumors can cause nausea and/or vomiting by increasing the ICP at the area postrema of the medulla. (See "Approach to the adult with nausea and vomiting").

Several characteristics suggest the possibility of tumor-associated emesis, such as triggering emesis by an abrupt change in body position. More importantly, neurogenic nausea and vomiting usually occur in the context of other neurologic symptoms such as headache or focal neurologic deficit; these signs and symptoms may be subtle.

**Syncope** — A significant rise in ICP can temporarily cut off cerebral perfusion, leading to loss of consciousness. Patients with brain tumors are particularly susceptible to this sequence of events in association with normal plateau waves.

Plateau waves are sustained pressure waves that normally occur within the brain and are caused by activities that transiently raise the ICP. As an example, any Valsalva maneuver (eg, coughing, sneezing, vomiting) that increases the intrathoracic pressure can impede jugular venous drainage, leading to a transient increase in ICP.

In the presence of a brain tumor, the baseline ICP may be raised to a level that reduces brain compliance; in this setting, even a further small increase in intracranial fluid volume can result in dramatic elevations in ICP. The cerebral perfusion pressure is the difference between the mean systemic arterial pressure and ICP. Thus, a significant rise in ICP can temporarily cut off cerebral perfusion, leading to loss of consciousness. (See "Evaluation and management of elevated intracranial pressure in adults").

A syncopal episode may simulate a seizure, since patients suffer loss of consciousness and may have a few tonic-clonic jerks. Identification of plateau wave-related episodes of loss of consciousness is critical, since these events identify patients who require urgent corticosteroids and/or neurosurgical intervention to reduce elevated ICP rather than treatment with an anticonvulsant. (See "Management of vasogenic edema in patients with primary and metastatic brain tumors", section on 'Glucocorticoids'.)

**Cognitive dysfunction** — Cognitive dysfunction, which includes memory problems and mood or personality change, is common among patients with intracranial malignancy.

Most of the neurocognitive deficits associated with brain tumors are subtle. Patients often complain of having low energy, fatigue, an urge to sleep, and loss of interest in everyday activities. They may become abulic and show a lack of spontaneity. This pattern of symptoms can be confused with depression. (See "Clinical manifestations and diagnosis of depression").

Because these symptoms lack specificity, they are often recognized in retrospect by the patient or the treating physician. Thus, consideration should be given to neuroimaging to rule out a brain tumor in patients without a prior history of depression who experience new onset of depressive symptoms without obvious cause.

**Focal**

**Weakness** — Muscle weakness is a common complaint in patients with brain tumors. The manifestations may be subtle, particularly in the early stages. For upper motor neuron lesions, weakness is generally more pronounced in the flexors of the lower extremities than in the extensors, and more pronounced in the extensors than in the flexors in the upper extremities. Transient weakness may represent a postictal state, as in Todd's paralysis (see 'Seizures' above). (See "The detailed neurologic examination in adults", section on 'Upper versus lower motor neuron lesions'.)

A key feature of tumor-related muscle weakness is its frequent responsiveness to high-dose dexamethasone, particularly with tumors that are near the motor cortex or its descending fibers. A response to dexamethasone usually means that the weakness is caused by edema and not by direct tumor involvement. In such a patient, craniotomy can be considered to relieve mass effect and lessen...
the corticosteroid requirement, even if the patient is not resectable for cure. (See "Management of vasogenic edema in patients with primary and metastatic brain tumors", section on 'Glucocorticoids'.)

**Sensory loss** — Cortical sensory deficits (eg, graphesthesia or abnormalities in stereognosis) can develop in patients whose tumors invade the primary sensory cortex (table 5). These sensory deficits usually do not respect a dermatomal or peripheral nerve distribution. (See "The detailed neurologic examination in adults", section on 'Sensory examination'.)

The type of deficit varies in part with location of the tumor. As an example, visual-spatial disconnection syndromes can be found in patients with tumors located in the visual association cortex (see 'Visual spatial dysfunction' below). Some of the other cortically-based sensory deficits include loss of spatial orientation, tingling, and lack of coordination.

**Aphasia** — Aphasia is a disorder of language function, not of vocalization, as in dysarthria or hoarseness. It is a specific sign of a lesion in the dominant hemisphere (usually left frontal or parietal); in comparison, lesions in the nondominant hemisphere may produce apraxia, which refers to an inability to perform purposeful movements (table 5).

Patients who are aphasic may be confused with those who have dementia or other psychiatric disorders such as psychosis or depression. Subacute psychosis in a patient without an antecedent psychiatric history is a diagnosis of exclusion that requires diagnostic neuroimaging studies to rule out an organic cause.

**Visual spatial dysfunction** — The visual pathway courses through the brain from the retina and optic chiasm to the occipital poles of the cerebral cortex. Because of its long course through the brain, the visual pathway can be affected by brain tumors that involve any of these areas.

- Examination of the retina, particularly the optic discs, is an important component of the initial examination of a patient suspected of having a brain tumor. The optic nerve head can be a good measure of ICP, although papilledema is often not present in the elderly despite an increase in ICP. Although gross papilledema is easy to diagnose, evolving papilledema can be difficult to recognize. As an example, only the medial portion of the optic disk may be blurred initially in early papilledema. Venous engorgement and the absence of venous pulsations are also important diagnostic clues. (See "Overview and differential diagnosis of papilledema").

- Tumor-related compression of the optic chiasm usually manifests as a bitemporal hemianopsia. However, there may be a unilateral hemianopsia with a central scotoma in the contralateral eye in the early stages, particularly when the compression is on one side. The latter results from partial compression of the Wilbrand's knee in the contralateral optic nerve. (See "Causes, presentation, and evaluation of sellar masses" and "Optic pathway glioma").

- Postchiasmatic lesions also may present with visual abnormalities. Because of the somatotopic representation of the visual pathways, the more anterior the lesion, the more asymmetric the hemianopsia.

**DIAGNOSTIC NEUROIMAGING** — The differential diagnosis of an adult presenting with signs and symptoms suggesting a brain tumor includes both neoplastic and nonneoplastic conditions (table 6). Neuroradiologic imaging is the major diagnostic modality in the evaluation of brain tumors. These studies are critical for preoperative planning, and they often provide information about the etiology of a mass lesion.

Although neuroimaging cannot definitively establish the specific histology, the characteristic appearance of dural-based meningiomas often can allow a tentative diagnosis. Treatment of meningiomas without a histologic diagnosis may be warranted in carefully selected cases for lesions in a surgically inaccessible location. (See "Meningioma: Clinical presentation and diagnosis", section on 'Neuroimaging'.)
**Magnetic resonance imaging** — Gadolinium-enhanced magnetic resonance imaging (MRI) is usually the only test needed to suggest a brain tumor. MRI may also provide information that indicates the specific tumor type:

- Malignant gliomas are typically hypointense on T1-weighted images, and enhance heterogeneously following contrast infusion. Enhancing tumor can be distinguished from the surrounding hypointense signal of edema on T1-weighted sequences (image 1).
- Regardless of the histologic grade, astrocytomas generally show increased T2 and FLAIR signal intensity; however, some astrocytomas do not manifest contrast enhancement [15].
- Low grade gliomas generally present as an infiltrating hemispheric lesion that produce little mass effect. The MRI appearance of focal brainstem gliomas is discussed elsewhere. (See "Focal brainstem glioma", section on 'Clinical presentation'.)

In addition to permitting visualization of the tumor and its relationship to the surrounding normal parenchyma, MRI is also superior to computed tomography (CT) for evaluation of the meninges, subarachnoid space, and posterior fossa, and for defining the vascular distribution of the abnormality. (See 'Computed tomography' below.)

**Magnetic resonance spectroscopy** — Magnetic resonance spectroscopy (MRS) is increasingly being utilized as a diagnostic technique in patients with suspected brain tumors [16-19]. This technique may improve the differentiation of locally infiltrative brain tumors from other types of well-circumscribed intracranial lesions by analyzing the chemical composition in an area of interest selected by the radiologist. Important spectroscopic signals include N-acetylaspartate, choline, lactate, and 2-hydroxyglutarate (table 7):

- N-acetylaspartate, a byproduct of the ubiquitous neurotransmitter glutamate, signals the presence of neurons since it localizes to the synaptic terminals, and is decreased in gliomas.
- Choline, a component of cell membranes, is increased in tumors.
- Lactate, a product of anaerobic respiration, can be present in necrotic tumor, infection, or stroke.
- Mutations in IDH1 and IDH2 result in accumulation of 2-hydroxyglutarate [20-22].

MRS cannot differentiate the types of infiltrative or circumscribed lesions, and it cannot replace histologic diagnosis of malignancy [18]. However, it can help differentiate between neoplasm and other central nervous system processes [23]. The utility of MRS was illustrated in a study of 26 patients with intracranial masses who underwent MRI, proton MRS, and stereotactic biopsy [18]. For patients with gliomas and lymphomas, pathologic spectra were present outside the area of contrast enhancement, suggesting infiltration beyond the borders suggested by MRI. In comparison, four circumscribed tumors (meningioma, pineocytoma, metastasis, and germinoma) showed no pathologic spectra outside the region of enhancement.

MRS spectroscopy signals are degraded by bone and cerebrospinal fluid. As a result, MRS is less useful for skull base and periventricular lesions.

**Functional MRI** — When a region of the brain is activated (eg, the language center during phonation or the motor cortex when moving a limb), blood flow to that region increases. Functional MRI (also known as echoplanar MRI) permits the measurement of differences in blood flow though particular regions of the brain and has several advantages in patients with brain tumors:

- Functional MRI is an important adjunct in the preoperative planning for patients whose tumor is located near eloquent areas of the brain [24]. In such areas, imaging after activation of sensory
and motor areas by appropriate stimuli may permit separation of tumor from normal brain preoperatively (functional brain imaging) [25,26].

- Functional MRI may permit better resolution of tumor versus surrounding edema at the tumor borders [27,28].
- Ultrafast acquisition of MRI images by echoplanar imaging is free from motion artifact and may be useful in patients who are unable to cooperate with standard MRI scanning [29].

**Perfusion MRI** — Perfusion MRI is the imaging of blood flow in brain tumors. This is done either with a bolus of gadolinium as in dynamic contrast MRI [30] or dynamic susceptibility contrast MRI [31], or with magnetic pulsation of water molecules as they pass through carotid and vertebral arteries as in arterial spin labeling [32,33]. Increased perfusion can be seen in newly diagnosed or recurrent brain tumors, usually reflecting the presence of hypervascularity.

**Computed tomography** — CT has largely been replaced by cranial MRI as the imaging modality of choice for brain tumors. However, CT retains utility in selected situations:

- To detect bone or vascular involvement
- To detect metastases to the skull base, clivus, or regions near the foramen magnum
- In an emergency situation (e.g., an unstable patient with a suspected intratumoral hemorrhage), when CT can be faster to perform than MRI
- In patients for whom MRI is contraindicated, either because of an iron-containing implant or because of claustrophobia

**PET scans** — Positron emission tomography (PET) with fluorodeoxyglucose (FDG) can be used to detect malignant tumors with high metabolic rates. Such lesions take up greater amounts of glucose than surrounding tissues or tumors with slower metabolic rates.

FDG-PET may be useful in the following situations:

- To localize the areas of maximum glucose utilization within the tumor. This information can guide the neurosurgeon to biopsy tumor locations with the most aggressive biologic behavior [34-36].
- To permit the mapping of functional areas of the brain prior to surgery or radiation, in conjunction with functional MRI, in order to minimize injury to eloquent areas [25,37,38].
- To differentiate recurrent tumor from radiation necrosis [39-43].
- To help differentiate high-grade (glucose hypermetabolizing) from low-grade (glucose hypometabolizing) brain tumors [44,45]. In patients with low-grade gliomas, FDG-PET images that show diffuse hypometabolism may support a decision to defer treatment, while the presence of hypermetabolic areas may indicate a high-grade tumor and signal the need for biopsy or treatment. (See "Diagnosis and classification of low-grade gliomas".)

Because of high basal uptake of FDG by normal brain tissues, the low signal-to-noise ratio of FDG-PET may result in false negative data [46], sensitivity of 71 percent and specificity of 80 percent [43]. As a result, FDG-PET is not an FDA-approved diagnostic study for evaluating malignancies in the brain, even though it is approved for systemic malignancies. 18F-fluorothymidine-PET (18F-FLT-PET) has a higher signal-to-noise radiation than FDG-PET [47]. But its exact utility in the brain tumor population await further clinical trials.

**SPECT imaging** — Single photon emission CT (SPECT) utilizes various isotopes to detect abnormalities in the blood brain barrier. Preoperative SPECT using thallium-201 is useful to distinguish...
benign from malignant brain lesions, predict the histologic grade of brain tumors, and select areas for biopsy [48]. The uptake of 201-Tl is unaffected by steroid treatment [49].

There appears to be a correlation between early and delayed tumor uptake of 201-Tl and the subsequent grade of the surgically resected tumor, allowing a distinction between low- and high-grade astrocytomas [49-52].

**DIAGNOSTIC NEUROSURGICAL INTERVENTION** — Accurate diagnosis of a brain tumor requires an adequate tissue sample for histologic study. This may be obtained by stereotactic biopsy or open surgery.

Corticosteroid use should be avoided prior to biopsy or surgery if either a primary central nervous system lymphoma or an infectious process is part of the differential diagnosis. (See 'CNS lymphomas' below.)

**Preoperative assessment** — The likelihood that a lesion is metastatic should be assessed prior to proceeding to biopsy. Brain metastases are more common than primary brain tumors. Although brain metastases usually present in the context of overt systemic disease, they can occur as the initial manifestation of a systemic malignancy.

If any aspect of the clinical or neurodiagnostic evaluation suggests that a brain tumor is a metastatic rather than a primary lesion, systemic evaluation, particularly of the thorax, should be carried. Neurosurgeical intervention in a patient with a presumed brain metastasis should balance the risks of the procedure versus the potential benefits (ie, tissue diagnosis, resection of a solitary lesion, relief of neurological symptoms). (See "Overview of the clinical manifestations, diagnosis, and management of patients with brain metastases").

**Surgical exploration or biopsy** — Technical advances have improved the safety of both surgical resection and stereotactic biopsy. Improved diagnostic neuroimaging permits better preoperative localization of the lesion and separation of the lesion from adjacent normal brain tissue, particularly in eloquent areas of the brain. Techniques such as intraoperative MRI facilitate improved surgical navigation for lesion biopsy and resection.

For most patients, tissue for histologic confirmation is obtained at the time of surgical exploration for resection. In those patients with large, nonresectable lesions or those whose lesions are in a critical location, stereotactic biopsy is an alternative. In some patients with a meningioma with a lesion that is either difficult or impossible to access, the characteristic appearance of a dural-based tumor may be sufficient to permit treatment. (See "Treatment of benign (WHO grade I) meningioma", section on 'RT alone for nonresectable meningiomas'.)

**Glial tumors** — For patients with primary glial tumors, maximum surgical resection is usually recommended. (See appropriate topic reviews).

There are two exceptions to this approach:

- **Low-grade glioma** — For patients with low-grade glial tumors, the role of maximal resection remains uncertain. Retrospective studies suggest that more extensive tumor resection is associated with delay in tumor progression and malignant degeneration, as well as improved survival. (See 'Management of low-grade glioma', section on 'Surgery'.)

  Immediate surgery is generally required for patients presenting with a large mass or extensive neurologic symptoms, both to establish the diagnosis and to debulk the tumor. However, for patients with a small tumor not creating a mass effect and only transient symptoms or tumors in eloquent cortex, careful observation may be an alternative to immediate surgery.

- **Diffuse pontine glioma** — As long as characteristic radiologic features are present, a tissue diagnosis of a pontine gliomas not required prior to treatment (algorithm 1) [53]. In equivocal
cases, the clinician must weigh the risks of a surgical biopsy versus the diagnostic uncertainty. (See "Diffuse pontine glioma".)

CNS lymphomas — Patients with primary central nervous system lymphoma are typically not offered surgical debulking, since the extent of surgical resection does not influence patient survival. Stereotactic biopsy is usually recommended, providing a high rate of positive tissue diagnosis with a low rate (<2 percent) of morbidity and mortality. Definitive treatment is usually chemotherapy, radiation, or a combination of these modalities. (See "Clinical presentation, pathologic features, and diagnosis of primary central nervous system lymphoma" and "Treatment and prognosis of primary central nervous system lymphoma").

Corticosteroids should be avoided whenever possible prior to biopsy or surgery if primary CNS lymphoma is part of the differential diagnosis. These agents are lymphocytotoxic. A single dose can alter the histopathologic evaluation, and a short course of treatment may cause the lymphoma to disappear temporarily.

Brain metastases — Biopsy is not necessary for patients with multiple brain metastases if there is a preexisting primary cancer known to have a propensity for brain metastases. If, however, a patient has a primary tumor that does not usually metastasize to the brain (eg, prostate cancer), there is no known primary, or neuroimaging is not typical for metastasis, biopsy may be indicated to define the etiology of a brain lesion. The importance of biopsy in such situations was illustrated in a randomized trial of surgery for the treatment of solitary brain metastases, in which 11 percent of patients had a second primary malignancy, inflammatory process, or infection rather than metastasis [54].

The management of patients with brain metastases is dependent upon the overall condition of the patient, as well as the number, size, and location of brain metastases. The management of patients with brain metastases is discussed separately. (See "Treatment of brain metastases in favorable prognosis patients" and "Treatment of brain metastases in poor prognosis patients".)

Neuropathology — Histologic examination of the biopsy specimen remains the most important component of the diagnostic evaluation of brain tumors. A smear or frozen section can be performed in the operating room for a preliminary interpretation of the histologic subtype. With this information, the neurosurgeon can make a decision whether or not to proceed with a more extensive resection.

A definitive diagnosis requires examination of the permanent tissue sections stained by hematoxylin and eosin. Important information may be derived from additional analyses that include the following:

- Proliferative index, using the MIB-1 monoclonal antibody
- Immunohistochemical stains
- Electron microscopy may be necessary to ascertain the correct diagnosis
- Molecular markers, including deletions of chromosome 1p/19q or the presence or absence of promoter methylation of the DNA repair enzyme O6-methylguanine-DNA methyltransferase (MGMT), isocitrate dehydrogenase (IDH), and p53

Caution should be exercised in interpreting the tissue diagnosis when the tissue is derived only from a biopsy specimen. This is particularly true for glial tumors, because regional heterogeneity and sampling error may underestimate the histologic grade of malignancy [55].

In patients suspected of having a primary central nervous system lymphoma, corticosteroid use should be avoided prior to biopsy, as these drugs are lymphocytotoxic and can affect the histopathologic evaluation. (See "Clinical presentation, pathologic features, and diagnosis of primary central nervous system lymphoma", section on 'Diagnosis'.)
INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topics (see "Patient information: Brain cancer (The Basics)"
- Beyond the Basics topics (see "Patient information: Primary low-grade glioma in adults (Beyond the Basics)" and "Patient information: High-grade glioma in adults (Beyond the Basics)" and "Patient information: Meningioma (Beyond the Basics)"

SUMMARY — Brain tumors can produce symptoms and signs by local brain invasion, compression of adjacent structures, and increased intracranial pressure (ICP). In addition to the histology of the tumor, the clinical manifestations are determined by the function of the involved areas of brain. The proper evaluation of the patient with a suspected brain tumor requires a detailed history, a comprehensive neurologic examination, and appropriate diagnostic neuroimaging studies.

Although newer imaging techniques often provide information suggesting a specific histology, an adequate tissue sample should generally be obtained, either at the time of neurosurgical resection or by stereotactic biopsy, to optimize treatment.

Use of UpToDate is subject to the Subscription and License Agreement.

REFERENCES


