In this work, the brain lesions that cause spontaneously hyperintense T1 signal on MRI were studied under seven categories. The first category includes lesions with hemorrhagic components, such as infarct, encephalitis, intraparenchymal hematoma, cortical contusion, diffuse axonal injury, subarachnoid hemorrhage, subdural and epidural hematoma, intraventricular hemorrhage, vascular malformation and aneurysm, and hemorrhagic neoplasm. The second category includes protein-containing lesions, such as colloid cyst, craniopharyngioma, Rathke's cleft cyst, and atypical epidermoid. The third category includes lesions with fatty components, such as lipoma, dermoid, and lipomatous meningioma. Lesions with calcification or ossification, such as endocrine-metabolic disorder, calcified neoplasm, infection, and dural osteoma, constitute the fourth category, whereas the fifth category includes lesions with other mineral accumulation, such as acquired hepato-cerebral degeneration and Wilson disease. The sixth category includes melanin-containing lesions, such as metastasis from melanoma and leptomeningeal melanosis. The last category is the miscellaneous group, which includes ectopic neurohypophysis, chronic stages of multiple sclerosis, and neurofibromatosis type I. The above-mentioned lesions are presented with their typical T1-hyperintense images, and the underlying reasons for those appearances in magnetic resonance imaging are discussed.

The brain lesions that cause spontaneous T1 shortening on magnetic resonance imaging (MRI) were studied under seven categories in this article. These categories have included lesions with hemorrhagic components, protein-containing lesions, fatty lesions, lesions with calcification or ossification, lesions with other mineral accumulation, melanin-containing lesions, and a miscellaneous group (Table 1).

The purpose of our study is to review, illustrate, and discuss the MRI findings of the brain lesions causing T1 shortening with their typical T1-hyperintense appearances. The role of MRI in evaluating such lesions has been emphasized.

Lesions with Hemorrhagic Components

Brain Infarcts

Infarction of brain tissue usually result from vascular occlusive diseases involving arteries or (very rarely) veins. The arteries may be occluded for a variety of reasons, most commonly because of atherosclerotic arterial disease, followed by cardiovasculogenic embolic occlusion, hypercoagulable states, arterial dissection, congenital anomalies, and neoplastic infiltration or constriction of the arteries. Although the arterial occlusion causes infarction of a specific vascular territory of the brain with the involvement of cortical gray matter and subcortical white matter, venous occlusions secondary to thrombophlebitis, hypercoagulable states, dehydration, oral contraceptive usage, and tumoral encasement lead to areas of infarction that do not correspond to arterial distributions and primarily affect the subcortical white matter rather than cortical gray matter.1-4

The MRI appearance of cerebral infarcts depends mainly on the age of infarct at the time of examination. Areas of T1 shortening often develop within the zones of subacute infarcts, starting on the second day. A ribbon of high-signal intensity on T1-weighted scans is seen following the contour of involved cerebral cortex. This gyriform pattern may be caused by petechial hemorrhage and biochemical changes caused by laminar necrosis, ie, protein denaturation (Fig 1). However, T1-hyperintense areas may be seen starting...
from the second day of infarct until the end of second month if there is an associated area of hemorrhage secondary to the dissolution of an embolus allowing a reperfusion hemorrhage or to anticoagulant treatment (Fig 2). Multifocal hemorrhagic infarcts suggest an embolic source, such as subacute bacterial endocarditis. It is important to remember that hemorrhage within cerebral infarcts may mimic other causes of atypical hematomas, such as hemorrhagic neoplasms. Venous pathologies, such as dural sinus thrombosis, should also be considered as the underlying reason of hemorrhagic cerebral infarction. Additional T2-weighted MRI sequences help to determine the exact stage of infarct. Diffusion-weighted images show restricted diffusion (high signal) in cerebral infarcts (Fig 1).1,3-9

**Infections**

Encephalitis is a diffuse inflammatory state of brain tissue and most commonly develops secondary to viral infections. Viral encephalitis is usually seen in immunocompromised patients. Herpes encephalitis is the most common sporadic encephalitis in temperate climates and is caused by herpes simplex type I. Other less-common viral agents are cytomegalovirus, human immunodeficiency virus (HIV-1), and varicella-zoster virus.10,11

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**TABLE 1.** The lesions of brain with spontaneous T1 hyperintense signal characteristics on MRI

<table>
<thead>
<tr>
<th>1. Hemorrhagic lesions</th>
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<tbody>
<tr>
<td>A. Infarcts: Subacute phase of arterial infarct associated with gyral hemorrhage and protein denaturation, embolic infarct associated with reperfusion, and venous infarct</td>
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<tr>
<td>B. Infections, eg, herpes simplex encephalitis, cytomegalovirus encephalitis, and HIV encephalitis</td>
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<tr>
<td>C. Intraparenchymal hematoma: Traumatic, profuse hemorrhagic infarct, hypertensive hematoma, secondary to aneurysms and vascular malformations, germinal matrix bleeding, amyloid angiopathy, coagulopathies, and blood dyscrasias</td>
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<tr>
<td>D. Cortical contusions</td>
</tr>
<tr>
<td>E. Diffuse axonal injuries</td>
</tr>
<tr>
<td>F. Subarachnoid hemorrhage: Traumatic, secondary to aneurysms and vascular malformations</td>
</tr>
<tr>
<td>G. Subdural and epidural hematoma</td>
</tr>
<tr>
<td>H. Intraventricular hemorrhage: Traumatic, secondary to aneurysms and vascular malformations, periventricular hematomas dissecting into ventricles, germinal matrix bleeding, amyloid angiopathy, coagulopathies, and blood dyscrasias</td>
</tr>
<tr>
<td>I. Vascular malformations and aneurysms associated with intra- or perilesional hemorrhage and/or thrombosis: AVM, cavernous malformation, aneurysm</td>
</tr>
<tr>
<td>J. Hemorrhagic neoplasms: Primary tumors such as pituitary adenomas, anaplastic astrocytoma, glioblastoma multiforme, and lymphoma (in immunocompromised patients); secondary tumors such as metastasis from melanoma, renal cell carcinoma, choriocarcinoma, brochogenic carcinoma, and thyroid carcinoma</td>
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<th>2. Protein-containing lesions</th>
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<tr>
<td>A. Colloid cyst of third ventricle</td>
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<td>B. Craniohypophyseal neoplasm</td>
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<td>C. Rathke’s cleft cyst</td>
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<td>D. Atypical epidermoid</td>
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<th>3. Fatty lesions</th>
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<tr>
<td>A. Lipoma: pericallosal, cisternal, and intraventricular</td>
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<td>B. Dermoid</td>
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<td>C. Lipomatous meningioma</td>
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<th>4. Calcified/ossified lesions</th>
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<tbody>
<tr>
<td>A. Endocrine-metabolic disorders: Hypo- or hyperparathyroidism, hypothyroidism, mitochondrial encephalopathies, Fahr disease (familial cerebrovascular ferrocacinosis), carbon monoxide poisoning, and idiopathic calcification</td>
</tr>
<tr>
<td>B. Calcified neoplasms: Craniohypophyseal neoplasm, oligodendroglioma, choroid plexus papilloma, meningioma, pituitary adenoma, astrocytoma, pericallosal lipomas, ependymomas, metastases from lung, breast, and gastrointestinal carcinomas</td>
</tr>
<tr>
<td>C. Infections: Toxoplasmosis, cytomegalovirus, rubella, herpes, tuberculosis, and cysticercosis infections</td>
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<tr>
<td>D. Dural osteomas</td>
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<th>5. Lesions with other mineral accumulation</th>
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<tr>
<td>A. Acquired hepatocerebral degeneration</td>
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<td>B. Wilson’s disease</td>
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<th>6. Melanin-containing lesions</th>
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<tr>
<td>A. Melanoma metastases</td>
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<td>B. Leptomeningeal melanosis</td>
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<th>7. Miscellaneous</th>
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<tr>
<td>A. Ectopic neurohypophysis</td>
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<td>B. Multiple sclerosis (chronic stage)</td>
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<td>C. Neurofibromatosis type I</td>
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Herpes simplex encephalitis causes an acute fulminating hemorrhagic and necrotic type of meningoencephalitis that typically begins in the medial anterior temporal and orbital surface of frontal lobes. As herpes encephalitis progresses, patchy areas of hemorrhage and abnormal contrast enhancement are common. Small hemorrhages within the edematous tissue may be apparent on MRI as zones of T1 and/or T2 shortening (Fig 3). A rare alternative cause of rapidly progressive edema and patchy hemorrhage in the temporal lobe is thrombosis of the transverse sinus with venous infarction in the patients with herpes encephalitis. Involvement of the insular cortex, cingulate gyrus, and white matter lateral to the lenticular nucleus is characteristic of herpes encephalitis. The involvement of cingulate gyrus and contralateral limbic system is highly suggestive for herpes encephalitis.10-13

Intraparenchymal Hematoma

Intraparenchymal hematoma may result from trauma, hemorrhagic transformation of cerebral infarct, hypertension, amyloid angiopathy, rupture of aneurysms or vascular malformations, germinal matrix bleeding, coagulopathy and blood dyscrasias, neoplastic lesions, and infectious lesions. Hypertension, amy-
loid angiopathy, and coagulopathy are the most common causes of nontraumatic intracranial hemorrhage in the elderly, whereas patients younger than 50 usually have underlying vascular malformations and aneurysms. Hypertensive bleeding is frequently observed in the region of the basal ganglia, thalami, and internal capsules (Fig 4). Lobar or subcortical location of hematomas is generally seen with amyloid angiopathy, vascular malformations, and aneurysms. Hemorrhagic transformation of cerebral infarcts is usually seen 24 to 48 hours after the initial formation of infarct. It has a common predilection for the basal ganglia (Fig 5) and the cortex, and deep hemorrhagic infarctions are often associated with proximal middle cerebral artery occlusion.\textsuperscript{11,14-21}

The signal-intensity characteristics of an intracranial hematoma depend on several factors, the most important of which are listed as the age, size, location, hemoglobin oxidation state of hematoma, degree of clot retraction, extent of edema around hematoma, and hematocrit level of the patient. The MRI appearance of intracerebral hemorrhage on T1-weighted scans changes during the first week of bleeding. Iron of hemoglobin in the +2 state becomes oxidized to the +3 state in methemoglobin, which is strongly para-

![Fig. 3. Spin-echo T1-weighted axial image of a 42-year-old male patient with herpes simplex encephalitis reveals areas of gyriform high-signal intensity along the anteromedial parts of left temporal lobe, representing the subacute phase of patchy hemorrhagic areas.](image)

![Fig. 4. Spin-echo T1-weighted (A) and gradient-echo T2-weighted (B) axial images of a 67-year-old male patient with a history of long-standing hypertension and a right-sided hemiparesis that started 5 days previously reveal a subacute stage of hematoma involving parts of head of caudate nucleus, internal capsule, and putamen with high-signal intensity on T1-weighted images and very low-signal intensity on T2*-weighted images (black arrows). There are areas of hemorrhages at the same age within the left-sided corpus of the lateral ventricle (white arrows) secondary to the dissection of parenchymal hematoma into the ventricular system.](image)
magnetic during the early subacute phase of bleeding that starts around the second day of bleeding. When methemoglobin is initially intracellular, the hematoma has a high signal on T1-weighted images that progresses from periphery to center and a low signal on T2-weighted images that is surrounded by a zone of high T2 signal secondary to edema. When methemoglobin eventually becomes primarily extracellular during the late subacute phase of bleeding that starts around the first to second week of bleeding, the hematoma has a high signal on both T1- and T2-weighted images. Cell lysis and watery dilution of blood products accompany the oxidation of intracerebral hematomas, progressing inward from the periphery of the lesion. The combination of these events converts the initially low-signal intensity of acute hematomas on T2-weighted scans to high-signal intensity over a period of weeks. The concentric zones of evolving signal intensity are typical of most spontaneous intracerebral hematomas.14-16

**Cortical Contusion**

Cortical contusions are the most common traumatic injuries of brain parenchyma. They are often superficial lesions, reflecting bruising of the cortical surface against the adjacent osseous ridge and, less often, a dural fold. Cortical contusions most frequently result from acceleration/deceleration forces; however, a direct cortical contusion area may develop adjacent to a skull fracture, as in the case of a depressed fracture. Common locations include the anteroinferior portions of temporal lobes, perisylvian cortex, anteroinferior temporal lobes, and (less commonly) occipital pole. Evidence of parenchymal damage usually should be sought both immediately beneath and directly opposite a skull fracture or scalp injury. Petechial cortical contusions tend to coalesce into larger foci of hemorrhage, and they often become more evident within 24 to 48 hours after the initial trauma. The appearance of contusion on MRI is an area of focal hemorrhage involving the cerebral cortex and subcortical white matter. The signal of cortical contusion depends on the factors related to the hematoma. As the edema and mass effect subside during the subacute phase, T1 shortening develops with a gyriform contour following the cortical convolutions. The ribbon-like distribution of subacute hemorrhage is commonly seen within zones of contusion, resembling the pattern of T1 shortening in subacute infarction or anoxia (Fig 6). Wide hemorrhagic areas may be associated with contusion in some severe cases.22-25

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**Fig. 5.** Spin-echo T1-weighted axial image of a 63-year-old male patient with a history of longstanding hypertension reveals a hemorrhagic transformation of right putaminal infarct in the distribution of lateral lenticulostriate branches of middle cerebral artery with the high-signal intensity starting from the periphery and progressing toward the center of infarct area (arrow).

**Fig. 6.** Spin-echo T1-weighted axial image of a 45-year-old male patient with a history of recent head trauma reveals a hemorrhagic area of cortical contusion involving right occipital pole with heterogeneous high-signal intensity (arrow).
Diffuse Axonal (Shearing) Injuries

Diffuse axonal injuries, or shearing injuries, constitute the second most common traumatic injury of the brain. Acceleration/deceleration injury, or shaking of the brain, causes impaired axoplasmic transport, axonal swelling, and disconnection; thus, a state of axonal transection. Diffuse axonal injury is frequently observed near interfaces between tissues of different consistency, so that shearing forces develop when rotation or acceleration/deceleration forces impart different velocities and momentum to parenchyma on each side of the tissue boundary. The most common locations of shearing injuries are gray–white matter junctions in the frontal and temporal lobes, corpus callosum, fornix, brainstem, basal ganglia, and internal capsule. The size of shearing injuries may range from a few millimeters to centimeters. The lesions are commonly microscopic and nonhemorrhagic; however, up to 50% of shearing injuries may be hemorrhagic (Fig 7).17,26-28

Subarachnoid Hemorrhage

Subarachnoid hemorrhage may result secondary to a variety of underlying reasons, such as the trauma, rupture of aneurysms (Fig 8), bleeding vascular malformations, hematomas dissecting into the cerebrospinal fluid, and coagulopathy. Subarachnoid hemorrhage is not usually seen on MRI unless there is profuse bleeding because the extravasated blood elements are diluted within cerebrospinal fluid. Computed tomography is recommended for diagnosis of subarachnoid hemorrhages. However, in the case of profuse bleeding into the subarachnoid space independent of the underlying reason, hemoglobin may be deoxidized to the deoxyhemoglobin state and further to the methemoglobin state, giving a hyperintense appearance on T1-weighted images. Chronic stages of subarachnoid bleeding may be characterized by the deposition of hemosiderin on the pial surface of the brain, leading to a thin hypointense rim appearance, especially prominent on T2-weighted images, called superficial siderosis.29,30

Epidural and Subdural Hematomas

Epidural hematomas usually result from traumatic rupture of an epidural artery, often the middle meningeal artery, or of a dural venous sinus. The blood is collected between the skull and outer layer of dura mater with a typical biconvex shape. They do not cross suture lines, except in cases of very large hematomas associated with a diastatic fracture. Epidural hematomas cause compression of underlying brain parenchyma and subarachnoid spaces, and herniation is commonly detected. Epidural hematomas are com-
monly associated with skull fractures but rarely can be seen without fractures.\textsuperscript{14,22-24,31}

Subdural hematomas usually result from the tearing of cortical arteries or veins where they enter the subdural space. They have a typical crescentic shape and are located between the inner layer of dura and arachnoid layer. Subdural hematomas may cross suture lines but not dural attachments. They usually extend into the interhemispheric fissure. More than two thirds of the subdural hematomas have significant associated lesions, such as subarachnoid hemorrhage, cortical contusion, and diffuse axonal injuries.\textsuperscript{14,22-24,32,33}

The signal intensity of epidural and subdural hematomas depends on several factors, but the most important factor is the age of the hematoma at the time of examination. Acute hematomas are isointense or slightly lower in signal intensity than cerebral parenchyma on T1-weighted images. The hematomas have a high-signal intensity on T1-weighted images during the whole subacute stage, whereas they have a low-signal intensity secondary to the presence of intracellular methemoglobin during the early phase of the subacute stage, and a high-signal intensity secondary to the presence of extracellular methemoglobin during the late phase on T2-weighted images (Fig 9). Mixed signal intensities may be observed, especially for subdural hematomas, as a result of rebleeding into the hematomatous area.\textsuperscript{14,16,31-33}

\textbf{Fig. 8.} Spin-echo T1-weighted axial images of a 41-year-old female patient who had a subarachnoid bleeding 3 days ago reveal (A) a saccular aneurysm with a signal void at the bifurcation of right middle cerebral artery (white arrow) that is surrounded by hyperintense subacute bleeding within the subarachnoid space (black arrow), and (B) bleeding filling the right sylvian fissure as well (black arrow).

\textbf{Fig. 9.} Spin-echo T1-weighted axial image of a 70-year-old female patient with a history of recent head trauma reveals a rightsided crescentic subdural hematoma along the temporo-occipital surface (black arrows) with a hyperintense signal characteristic, representing subacute phase of hematoma. Note that an additional focal area of subdural hematoma is present along the right occipital pole (arrowhead).
Intraventricular Hemorrhage

Intraventricular hemorrhage may be seen as a result of several conditions, such as trauma, bleeding secondary to vascular malformations and aneurysms, periventricular hematomas dissecting into ventricles (Fig 4), germinal matrix bleeding (Fig 10), amylod angiopathy, coagulopathies, and blood dyscrasias. Hemorrhage commonly arises from fragile vessels in the periventricular germinal matrix of premature newborns. Hematomas may remain confined to the subependymal region or rupture into the ventricular system, often causing secondary hydrocephalus. In adults, hematomas in the posterior fossa or basal ganglia may rupture into the fourth, third, or lateral ventricles. The ventricles may be grossly expanded by thrombus.14,17,19,21,23,30,34-36

Vascular Malformations and Aneurysms

Arteriovenous malformations (AVMs) are the most common symptomatic cerebral vascular malformations. AVMs may be sporadic, congenital, or associated with a history of trauma. They are angiogenically immature lesions with continuing vascular remodeling. They are most commonly located in the supratentorial area (80-90%), whereas an infratentorial location is rare. AVMs contain multiple tightly packed tortuous tubular flow void structures on T1-and T2-weighted images secondary to patent arteries with high blood flow. They may contain thrombosed vessels with variable signal, areas of hemorrhage in various phases, calcification, and gliosis as well. The signal-intensity characteristics of AVMs depend on the presence of various components, as mentioned previously. Areas of T1 shortening may be detected secondary to thrombosis, hemorrhage, or calcification. AVMs do not contain brain tissue within the nidus of lesion. They are not usually associated with mass effect, except in the case of a recent hemorrhage or venous occlusion.14,16,19

Cavernous malformations, or cavernomas, are the most common angiographically occult vascular malformations. They are angiogenically immature dynamic lesions with endothelial proliferation and increased neangiogenesis. Almost 75% of the cavernomas occur as solitary sporadic lesions, whereas the rest of lesions are detected in the multiple familial form. They are collections of sinusoidal vascular spaces surrounded by a gliotic pseudocapsule without intervening neuroglial tissue. MRI appearance of cavernous angiomas is highly characteristic. An aggregate, multinodular, or popcorn morphology with prominent central zones of T1 shortening is surrounded by a characteristic rind of T2 shortening that is attributable to the accumulation of hemosiderin from old hemorrhages (Fig 11). Calcification may also contribute to low-signal intensity within and surrounding cavernous angiomas. They may range in size from a few millimeters to several centimeters in diameter. Any region of the brain may be affected; however, supratentorial cavernomas occur more frequently than infratentorial lesions. They have a propensity for repeated intraventricular hemorrhage.14,16,20,37,38

Intracranial aneurysms constitute the most common underlying reason for nontraumatic subarachnoid hemorrhage, and they may cause areas of T1 shorte-
ing during the subacute phase of subarachnoid bleeding, as explained above. The aneurysms may be saccular, fusiform, or blister-like structures. Saccular aneurysms occur typically at arterial bifurcations, and they are frequently located around the circle of Willis. Saccular aneurysms greater than 25 mm in diameter are referred to as giant aneurysms. The lumina of giant saccular aneurysms may be at least partially occupied by a thrombus that often has a lamellar or concentric appearance. The layers of intermediate- and high-signal intensity caused by thrombi of different ages and an additional zone of signal void layer representing the patent lumen are seen. This laminated internal architecture is a hallmark of thrombosed aneurysm. The circulation of blood within large-sized aneurysms is often complex, with lower flow velocities and less turbulence than is typically present within the lumen of smaller aneurysms. As a result, large aneurysms may demonstrate mixed intraluminal signal intensity on MRI scans. It may be difficult to distinguish between slowly flowing but patent components of the aneurysm and intraluminal thrombus on standard spin-echo scans. Flow-sensitive sequences are important in evaluating such cases.14,29,30,39,40

Hemorrhagic Neoplasms

Intracranial neoplasms, whether primary or metastatic, may be associated with hemorrhagic components. The reported incidence of hemorrhage in intracranial neoplasms varies from 1% to 15% in various series. Higher grades of malignancy, presence of neovascularity, presence of arteriovenous shunts within the lesions, rapidly growing neoplasms with associated necrosis, and vascular invasion by neoplasms may be the underlying factors contributing to the bleeding within the neoplasms. Primary tumors, such as pituitary adenomas, anaplastic astrocytoma, oligodendroglioma, glioblastoma multiforme, and lymphoma (in the case of immunocompromised pa-
tients), and secondary tumors, such as metastasis from bronchogenic carcinoma, renal cell carcinoma, melanoma, choriocarcinoma, and thyroid carcinoma, are the most common intracranial neoplasms associated with hemorrhage.21,41-44

The MRI appearance of a hemorrhagic neoplastic lesions is usually characterized by a more complex heterogeneous pattern of bleeding, nonhemorrhagic components that enhance after intravenous gadolinium administration, disordered evolution of hemorrhagic elements within the lesion, persisting peripheral edema during the evolution of lesion, and mass effect. Primary tumors are solitary, and metastatic tumors are typically multiple.21,43,44

Pituitary adenomas are common, benign, slowly growing tumors representing approximately half of the sellar and parasellar neoplasms in adults. Pituitary adenomas commonly have intermediate signal-intensity characteristics on T1- and T2-weighted sequences. However, the cysts, hemorrhage, and necrosis, which are more common in the case of macroadenomas with a size of more 10 mm, may be associated with adenomas, leading to a heterogeneous signal-intensity pattern (Fig 12). The pattern of hemorrhage within pituitary adenomas varies from homogeneous to heterogeneous. The hemorrhages within an adenoma are much more commonly microscopic and subclinical. Acute bleeding into an adenoma may cause sudden enlargement of the tumor, with compression on the optic chiasm and on the rest of hypophyseal gland. Rapidly developing visual impairment and endocrine dysfunction, usually accompanied by headache, is

Fig. 13. Spin-echo T1-weighted axial [A] and FLAIR coronal [B] images of a 52-year-old male patient with a left frontal opercular mass involving cortical gray matter and subcortical white matter that has a high-signal intensity after gyral convolutions on T1-weighted image [arrow] and an almost homogeneous high-signal intensity [arrow] on FLAIR image with associated profuse edema. The mass was surgically proven to be an oligodendroglioma with hemorrhagic components.

Fig. 14. Spin-echo T1-weighted axial image of a 48-year-old female patient with a right occipitotemporal mass lesion that has a peripheral high-signal intensity (arrowheads) toward the cortical surface of mass. The diagnosis was confirmed as anaplastic astrocytoma with hemorrhagic components at surgery.
called pituitary apoplexy. Some observers suggest an increased incidence of pituitary hemorrhage in patients receiving bromocriptine therapy.43,45-47

Anaplastic astrocytoma, oligodendroglioma, glioblastoma multiforme, and lymphoma (in immunocompro-

Fig. 15. Spin-echo T1-weighted axial (A) and postgadolinium spin-echo T1-weighted axial (B) images of a 35-year-old female patient reveal typical butterfly appearance of a bifrontal mass that extends through the genu of corpus callosum. The mass has a high-signal intensity peripherally representing surgically proven hemorrhagic components, and it shows a strong peripheral enhancement after gadolinium administration.

Fig. 16. Spin-echo T1-weighted axial (A) and gradient-echo T2-weighted axial (B) images of a 61-year-old male patient with a history of surgical excision of malignant melanoma in his arm reveal a nodular lesion of the left periventricular area with peripherally hyperintense and centrally hypointense components on T1-weighted image (arrow). The lesion is strongly hypointense on T2*-weighted image, representing its hemorrhagic nature (arrow). The single focus was surgically removed, and the histologic examination confirmed the diagnosis of melanotic malignant melanoma.
Mised patients) are the other primary neoplasms associated with hemorrhage. Macroscopic hemorrhage is relatively uncommon in glial tumors. Microscopic hemorrhage is a common pathological feature within high-grade gliomas. Oligodendrogliomas are relatively rare, slowly growing gliomas with usually mixed histologic patterns. They make up a disproportionate share of lesions presenting with gross hemorrhage (Fig 13). Areas of signal void are also seen at sites of clump-like calcification. High-grade gliomas, including anaplastic astrocytoma (Fig 14) and glioblastoma multiforme (Fig 15), are highly malignant neoplasms with necrosis and neovascular proliferation that represent a likely source of bleeding. Hemorrhage and necrosis also may be associated with primary brain lymphoma in immunocompromised patients.21,43-45

Metastases represent approximately one third of intracranial tumors. They are usually located at the gray–white matter interface of cerebral hemispheres. Hemorrhage, calcifications, and cysts may be associated with metastatic tumors. Neoplasms, such as melanoma (Fig 16), choriocarcinoma, hypernephroma, bronchogenic carcinoma (Fig 17), and thyroid carcinoma, usually cause hemorrhagic cerebral metastases. The high-signal intensity of T1-weighted images suggest the presence of subacute or early chronic hemorrhagic elements within the lesion. Hemorrhagic metastases may simulate the appearance of cavernous angiomas or other occult vascular malformations when they are solitary.21,43,44
Protein-containing Lesions

Colloid Cysts

Colloid cysts exclusively arise from the inferior aspect of the septum pellucidum and protrude into the anterior portion of the third ventricle between columns of the fornix; however, some authors have reported the presence of colloid cysts in unusual locations, such as the posterior fossa and lateral ventricles. Typical clinical presentation is with episodes of positional headaches caused by intermittent hydrocephalus. The third ventricle is enlarged to accommodate the cyst; lateral ventricles are enlarged as the result of obstruction in foramen of Monro. The cysts are well-circumscribed, with hyperintense signal-intensity on both T1- and T2-weighted sequences in almost 60% of the patients due to protein and mucin content of the lesion; however, they show variable signal-intensity characteristics in the rest of the patients, depending on the content of the cyst (Fig 18). Thin rim enhancement may be observed around the cyst, representing a fibrous capsule, after intravenous gadolinium administration. Differential diagnosis from the masses, such as meningioma and ependymoma, is not difficult because of their typical features on MRI.

Craniopharyngioma

Craniopharyngiomas are usually histologically benign but locally aggressive lesions arising from squamous epithelial rests along Rathke’s cleft. They may present with a variety of signal intensities, sizes, and morphologies on MRI pictures. Craniopharyngiomas most commonly involve the suprasellar region, alone or in combination with intrasellar components. The midline location is typical. They are usually well-circumscribed lobulated lesions with variable low-, intermediate-, or high-signal on T1- and T2-weighted images. T1-hyperintense components within craniopharyngiomas most often represent high protein content within cystic regions of the tumor. Experimental studies and analysis of fluid from tumor cysts have suggested that appreciable T1 shortening is usually correlated with protein concentrations in the range of 10% to 30%. Calcification and hemorrhage may be associated with craniopharyngiomas, causing variable signal-intensity patterns on T1- and T2-weighted images.

Rathke’s Cleft Cyst

Rathke’s cleft cysts are uncommon, benign cystic lesions that are derived from the remnants of the epithelium embryologically lining Rathke’s cleft (cranioopharyngeal duct). They may be intrasellar in 50% of the cases, suprasellar in 25% of the cases, and both in 25% of the cases. The cysts are usually simple, lined by a single epithelial layer. They may contain variable amounts of protein, mucopolysaccharide, cellular debris, and cholesterol. Their signal intensity may be
correspondingly high, low, or intermediate on T1- and T2-weighted sequences, depending on the contents of cystic lesions (Fig 19). Gadolinium enhancement is usually absent.47,52

**Atypical Epidermoid Cysts**

Epidermoid cysts are nonneoplastic, congenital, or acquired extra-axial off-midline lesions that are filled with desquamated cells and keratinaceous debris. The prepontine and cerebellopontine angle

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**Fig. 22.** Spin-echo T1-weighted sagittal image (A) and fat-suppressed spin-echo T1-weighted axial image (B) of a 34-year-old female patient show a well-circumscribed hyperintense mass (arrow) of the quadrigeminal cistern on T1-weighted image with a signal-void region around the lipomatous mass due to chemical shift artifact, and fat-suppressed image reveals that the hyperintense signal of the quadrigeminal cistern lipoma ceases totally (arrow).

**Fig. 23.** Spin-echo T1-weighted sagittal image of a 46-year-old male patient with headache reveals a hyperintense lipoma along the floor of hypothalamus in the suprasellar cistern (arrow).

**Fig. 24.** Spin-echo T1-weighted sagittal image of a 28-year-old female patient shows an incidentally detected subependymal mass with high-signal intensity (arrow) that ceased on fat-suppressed images. The diagnosis is the atrial lipoma.
cisterns and suprasellar and parasellar areas are common sites for epidermoid cysts. They are well-circumscribed, spheroid, or multilobulated cystic lesions. They usually cause encasement rather than displacement of the neighboring cranial nerves and vessels. They frequently have signal intensities similar to that of cerebrospinal fluid on MRI. Very rarely, epidermoid cysts demonstrate short T1 and T2 values resembling dermoid cysts (Fig 20). Such masses usually contain viscous liquid components containing relatively high concentrations of protein and causing high-signal intensity on T1-weighted scans. The lack of contrast enhancement is characteristic of epidermoid cysts.53-57

Fatty Lesions

Lipomas

Intracranial lipomas are uncommon congenital malformations that result from persistence and abnormal differentiation of the meninx primitiva. Microscopically, they are composed of adipose tissue with a variable mixture of vascular elements, musculo-collagenous fibers, and glial and ganglion cells. They are located at or near the midline in 80% to 95% of the cases. The common locations for lipomas are as pericallosal (25-50%) (Fig 21), sylvian fissure, quadrigeminal cistern (Fig 22), interpeduncular cistern, cerebellopontine angle cistern, cerebellomedullary cistern, chiasmatic-suprasellar cistern (Fig 23), and choroid plexus of the atrium (Fig 24). Lipomas can be nodular or curvilinear. They may contain calcifications. Intracranial lipomas are often asymptomatic and discovered...
incidentally. The clinical findings, if present, are usually related to associated anomalies, such as corpus callosum dysgenesis (Fig 21). Lipomas are characteristically hyperintense masses on T1- and less hyperintense masses on T2-weighted sequences, with decreased signal intensity on fat-suppressed sequences on MRI. A more specific clue to the diagnosis of lipoma and other lipid-containing masses is the chemical shift artifact, which is simply
related to the difference in resonant frequencies between fat and water protons. This artifact is displayed as a region of signal void at fat–water interfaces and hyperintensity at water–fat interfaces, along the frequency encoding axis.57-61

Dermoid Cysts

Dermoid cysts are benign, slowly growing cystic lesions arising from ectodermal cells enclosed intracranially during embryogenesis that are filled with lipid material, cholesterol, desquamated cells, and keratinaceous debris. Dermoid cysts may contain skin appendages, such as hair follicles, sweat glands, and sebaceous glands, in addition to squamous epithelium. Calcification is rare. They are found most commonly in the parasellar region. They are well-circumscribed extra-axial lesions with high signal on T1-weighted images and variable signal intensity on T2-weighted images. The lipid content of dermoid cysts causes high-signal intensity on T1-weighted scans (Fig 25). The fluid–fluid or fluid–debris levels may be present within the cystic lesions. They do not enhance after gadolinium administration. Dermoid cysts may leak or rupture, releasing their content into the subarachnoid space and ventricular system. The resultant appearance of scattered lipid droplets within sulci and cisterns is seen (Fig 26). Some patients may experience chemical meningitis, particularly when the contents of a ruptured cyst extend beyond the ventricles to involve the subarachnoid spaces. The rupture of dermoid cysts may even cause noncommunicating hydrocephalus.53,57,62,63

Lipomatous Meningioma

Meningiomas are the most common intracranial extra-axial tumors. They are usually benign, slowly
growing neoplasms; however, they may rarely reveal malignant behavior. They are well-circumscribed, dural-based lesions. They may cause compression of adjacent brain parenchyma, encasement of arteries, and compression of dural venous sinuses. The most common locations for meningiomas are the parasagittal area, convexity, sphenoid ridge, parasellar area, posterior fossa, optic nerve sheath, and intraventricular region. They are usually isointense with cortical gray matter on all MRI sequences with a prominent gadolinium enhancement. Calcification is a frequently detected characteristic of the lesions. Necrosis, cysts, and hemorrhage may be associated in around 25% of the masses. Dural tail reaction that is characterized by thickened dura tapering away from the tumor is characteristic, but not pathognomonic, for meningiomas.55,64,65

Lipomatous meningiomas are relatively rare, benign tumors that are characterized either by an admixture of mature adipocytes and meningioma or the production of lipids by neoplastic meningothelial cells assuming a lipoblast-like appearance. The lipomatous changes are thought to appear as a result of metaplastic changes of meningothelial cells. The signal intensity of lipomatous meningiomas is heterogeneous, but hyperintense components are detected on both T1- and T2-weighted images secondary to the presence of lipid (Fig 27).64-67

Fig. 31. Spin-echo T1-weighted axial image of a 38-year-old male patient who presented in a stuporous state with hepatic cirrhosis and high serum level of ammonium reveals symmetrical high signal intensity of bilateral globus pallidi (arrows). Additional high signal-intensity areas in the cerebral peduncles, dentate nuclei of cerebellum with the clinical findings of patient suggested the diagnosis of acquired hepatocerebral degeneration.

Fig. 32. Spin-echo T1-weighted axial image (A) and sagittal image (B) of 13-year-old female patient with the diagnosis of Wilson’s disease show symmetrical high-signal intensity characteristics of bilateral globus pallidi and nigrostriatal pathways (arrows).
Lesions with Calcified or Ossified Components

Endocrine–Metabolic Disorders

The signal intensity of calcification is usually characterized by hypointense appearance in both T1- and T2-weighted sequences on MRI. However, the presence of diamagnetic calcium salts and association with other paramagnetic cations, such as iron and manganese, may paradoxically cause T1 shortening in some cases. The size and configuration of calcium deposits are also important determinants of the signal intensity of calcification. Hypo- or hyperparathyroidism, hypothyroidism, mitochondrial encephalopathies, Fahr disease (familial cerebrovascular ferrocacinosis or bilateral striopallidodentate calcinosis) (Fig 28), carbon monoxide poisoning, and idiopathic calcification are commonly presented with intracranial calcifications. 68-72

Calcified Neoplasms

Primary neoplasms, such as craniopharyngioma, oligodendroglialoma (Fig 29), choroid plexus papilloma, meningioma, pituitary adenoma, astrocytoma, pericallosal lipoma, ependymoma, and metastases from lung, breast, and gastrointestinal carcinomas may contain areas of calcification. 47,72-78

Infections

Previous inflammatory diseases developing secondary to toxoplasmosis, cytomegalovirus, rubella, her-
pes, tuberculosis, and cysticercosis infections may cause foci of calcifications.\textsuperscript{10,68,70}

**Dural Osteomas**

Osteomas are benign proliferations of bone located in the skull or paranasal sinuses. The dural osteomas are very rarely detected lesions. The signal intensity of dural osteomas is characterized by a hyperintense appearance on T1- and T1-weighted images, probably secondary to fibro-fatty stromal components (Fig 30).\textsuperscript{79-81}

**Lesions with Other Mineral Accumulation**

**Acquired Hepatocerebral Degeneration**

Acquired hepatocerebral degeneration is a rare, nonhereditary, irreversible neurologic syndrome that occurs in patients with chronic liver disease associated with multiple metabolic insults. Most of the disorders of cerebral function develop rapidly and result mainly in a disturbance of consciousness ranging from confusion to stupor to coma and death. The pathophysiology and the locations of the cerebral injuries are incompletely understood. Enlargement and hyperplasia of protoplasmic astrocytes in the cerebral and cerebellar cortex, basal ganglia, and diencephalic nuclei are seen in acute forms of hepatocerebral degeneration. Gray and white matter necrosis with cavitation, gliosis, and myelin degeneration may occur in chronic forms. A variety of different MRI findings have been reported in these patients. Hyperintense signal changes on T1-weighted sequences and hypointense or isointense signal changes on T2-weighted sequences have been reported in the basal ganglia, subthalamic area, quadrigeminal plate, cerebral peduncles, internal capsules, and anterior pituitary gland (Fig 31). Bilateral symmetrical hyperintensity of the globus pallidus particularly is reported in T1-weighted images in 52% to 100% of patients with chronic liver diseases. This hyperintense signal change on T1-weighted images is reported to be caused by the deposition of manganese, most probably reflecting the presence of an adaptive process designed to improve the efficacy of ammonia detoxification by astrocytes. The signal hyperintensity may reverse after liver transplantation.\textsuperscript{82-84}

**Wilson’s Disease**

Wilson’s disease is an autosomal-recessive disease that is characterized by decreased functional serum ceruloplasmin levels and altered copper metabolism with increased urinary excretion of copper. The disease usually presents in childhood with abnormal toxic copper deposition in tissues, resulting in cirrhosis, and degenerative changes in the basal ganglia and brainstem. The signal-intensity changes on MRI are similar to those of acquired hepatocerebral degeneration (Fig 32).\textsuperscript{85-87}

**Melanin-containing Lesions**

**Melanoma**

Melanoma develops from melanocytes that are derived from neural crest cells. Melanoma typically causes hemorrhagic intracranial metastases (Fig 16); however, cerebral metastases may demonstrate hyperintense appearances on T1-weighted images and almost isointense to gray-matter appearance on T2-weighted images secondary to melanotic melanoma, even in the absence of hemorrhage (Fig 33). The presence of melanin molecules within the melanocytes with its unpaired electrons is believed to account for
the observed paramagnetic effects on signal intensity. Amelanotic metastases from primary melanomas are not associated with shortening of T1 or T2 values in the absence of hemorrhage.\textsuperscript{43,72,88}

**Neurocutaneous Melanosis**

Neurocutaneous melanosis is a rare neuroectodermal dysplasia that is characterized by the proliferation of melanocytes in leptomeninges associated with multiple congenital pigmented or giant hairy cutaneous nevi. It may change into primary central nervous system melanoma. The patients reveal multiple meningeal-based lesions usually less than 3 cm in diameter with intermediate- to high-signal intensity on T1-weighted images and variable signal intensity on T2-weighted images. The lesions reveal strong gadolinium enhancement. Vermian hypoplasia, arachnoid cysts, and Dandy–Walker malformation may be seen in association with neurocutaneous melanosis.\textsuperscript{88-90}

**Miscellaneous Lesions**

**Ectopic Neurohypophysis**

The normal neurohypophysis demonstrates high-signal intensity on T1-weighted images. T1 shortening of the neurohypophysis seems to reflect enhanced relaxation of water protons in the vicinity of neurosecretory vesicles that function in the storage and secretion of oxytocin and vasopressin. On sagittal MRI, the neurohypophyseal focus of T1 shortening lies immediately anterior to the dorsum sella and has been referred to as the posterior pituitary bright spot. The bright spot can be seen as an ectopic zone of T1 shortening representing the functional neurohypophysis along the floor of the third ventricle (Fig 34). This bright spot fails to develop in the normal intrasellar location because of impaired formation of the pituitary infundibulum, which normally transmits carrier-bound neuropeptide hormones from the hypothalamus to the neurohypophysis. The failure of development of the pituitary infundibulum and hypoplasia of the adeno-
hypothalamus may be isolated abnormalities or part of a midline dysgenesis syndrome.47,91,92

Chronic Phase of Multiple Sclerosis

Multiple sclerosis is the most common demyelinating disease. The plaques lesions of multiple sclerosis have usually low-to-intermediate-signal intensity on T1-weighted images and high-signal intensity on T2-weighted images with or without gadolinium enhancement depending on the activity of disease. Chronic plaque lesions of multiple sclerosis may be characterized by peripheral T1 shortening (Fig 35). The basis for such peripheral T1 shortening is undetermined but may be related to the accumulation of myelin degradation products. The finding is normally noted in the context of long-standing disease. Long-standing multiple sclerosis may also be associated with ventricular and sulcal enlargement, associated with variable degrees of cerebral atrophy.93-97

Neurofibromatosis Type I

Neurofibromatosis type I is the most common neurocutaneous syndrome with an autosomal-dominant transmission. Focal areas of high-signal intensity are present in the basal ganglia (most commonly globus pallidi), thalami, hippocampal gyri, and brainstem. The lesions are benign, multiple, nonenhancing signal areas with minimal or no mass effect. They are thought to represent hamartomas, spongiotic, or vascular changes of myelin with usually high signal on T2-weighted images and intermediate to slightly high signal on T1-weighted images (Fig 36). Although the precise reason for the signal-intensity changes is not clear, it may be attributed to an admixture of areas of myelin breakdown and remyelination of initially destroyed myelin and probable association of microcalcifications. They may show spontaneous regression over time by serial MRI.98-101

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