Brain MRI VINTON ALBERS, DC DRVALBERS.COM

FLAIR Fluid-Attenuated Inversion Recovery
Used to suppress the signal from water (CSF).
Suppression of fluid signals allows enhanced visualization of lesions.

Diffusion-Weighted Imaging (DWI)
Following an ischemic stroke, there is increased signal on a DWI scan. Water diffusion is restricted as a result of cytotoxic edema (cellular swelling).
The DWI enhancement appears within 5-10 minutes of the onset of stroke symptoms.

MR ANGIOGRAPHY- MRA
MRA capitalizes on creating intensity differences between flowing tissue and stationary tissue. By suppressing background stationary tissue and focusing only on the high-signal flowing blood, one can obtain a data set that depicts only vascular structures. If employed with contiguous sections or three-dimensional volumetric acquisitions, one can produce very thin section MR angiograms that can be rotated in space to visualize the circle of Willis or the carotid bifurcation. MR is capable of detecting atherosclerotic narrowings or intracranial aneurysms in three-dimensional space.

Ventricles/Cerebrospinal Fluid (CSF)
Flow of CSF is unidirectional: Lateral ventricles → interventricular foramina of Monro → third ventricle → cerebral aqueduct → fourth ventricle →foramina of Luschka and foramen of Magendie →cisterna magna (subarachnoid space) →arachnoid granulation →superior sagittal sinus. Volume of CSF: 70-160 mL. Normally, CSF is produced at the rate of 0.3cc/min (or 400-500 cc/day). CSF pressure: 80 to 180 mm H_20.

Epidural and Subdural Spaces
Epidural space: Between bone and dura, potential space in brain, but actual space in spinal cord.
Epidural hematoma: Accumulation of blood in the epidural space. Shape is convex lens. Usually see associated skull fracture due to torn meningeal vessel (usually middle meningeal artery). Uncontrolled arterial bleeding may lead to compression of the brain and subsequent herniation.

Subdural space: Between dura and arachnoid, potential space.
Subdural hematoma: Accumulation of blood in the subdural space. Shape is concave lens.

Subarachnoid space: Between arachnoid and pia, actual space.
Subarachnoid hemorrhage: Bleed into the subarachnoid space due to ruptured aneurysms or vascular malformations. Berry aneurysm (congenital). Mycotic aneurysm (infection). Vascular malformations.

CONCUSSION

A concussion is a traumatic brain injury that alters the way your brain functions. Effects are usually temporary, but can include problems with headache, concentration, memory, judgment, balance and coordination.

Although concussions usually are caused by a blow to the head, they can also occur when the head and upper body are violently shaken. These injuries can cause a loss of consciousness, but most concussions do not. Because of this, some people have concussions and don't realize it.

Concussions are common, particularly if you play a contact sport, such as football. But every concussion injures your brain to some extent. This injury needs time and rest to heal properly. Luckily, most concussive traumatic brain injuries are mild, and people usually recover fully.

Symptoms

The signs and symptoms of a concussion can be subtle and may not be immediately apparent. Symptoms can last for days, weeks or even longer.

The most common symptoms after a concussive traumatic brain injury are headache, amnesia and confusion. The amnesia, which may or may not be preceded by a loss of consciousness, almost always involves the loss of memory of the impact that caused the concussion.

Signs and symptoms of a concussion may include:

Headache or a feeling of pressure in the head

- Temporary loss of consciousness
- Confusion or feeling as if in a fog
- Amnesia surrounding the traumatic event
- Dizziness or "seeing stars"
- Ringing in the ears
- Nausea or vomiting
- Slurred speech
- Fatigue

Some symptoms of concussions may be immediate or delayed in onset by hours or days after injury:

- Concentration and memory complaints
- Irritability and other personality changes
- Sensitivity to light and noise
- Sleep disturbances
- Psychological adjustment problems and depression

- Disorders of taste and smell

Symptoms in children

Head trauma is very common in young children. But concussions can be difficult to recognize in infants and toddlers because they can't readily communicate how they feel. Nonverbal clues of a concussion may include:

- Listlessness, tiring easily
- Irritability, crankiness
- Change in eating or sleeping patterns
- Lack of interest in favorite toys
- Loss of balance, unsteady walking

Seek emergency care for a child who experiences a head injury and:

- Vomiting
- A headache that gets worse over time
- Changes in his or her behavior, including irritability or fussiness
- Changes in physical coordination, including stumbling or clumsiness
- Confusion or disorientation
- Slurred speech or other changes in speech
- Vision or eye disturbances, including pupils that are bigger than normal (dilated pupils) or pupils of unequal sizes
- Changes in breathing pattern
- Lasting or recurrent dizziness
- Blood or fluid discharge from the nose or ears
- Large head bumps or bruises on areas other than the forehead, especially in infants under 12 months of age

Seek emergency care for anyone who experiences a head injury and:

- A loss of consciousness lasting more than a minute
- Repeated vomiting
- Seizures
- Obvious difficulty with mental function or physical coordination
- Symptoms that worsen over time

Athletes

No one should return to play or vigorous activity while signs or symptoms of a concussion are present. Experts recommend that an athlete with a suspected concussion not return to play until he or she has been medically evaluated. Experts also recommend that child and adolescent athletes with a concussion not return to play on the same day as the injury.

Causes

Your brain has the consistency of gelatin. It's cushioned from everyday jolts and bumps by the cerebrospinal fluid that it floats in, inside your skull. A violent blow to your head

and neck or upper body can cause your brain to slide back and forth forcefully against the inner wall of your skull. Sudden acceleration or deceleration of the head — resulting from events such as a car crash or being violently shaken (shaken baby syndrome) — also can cause brain injury.

These injuries affect brain function, usually for a brief period, resulting in signs and symptoms of concussion. A brain injury of this sort may even lead to bleeding in or around your brain causing symptoms, such as prolonged drowsiness and confusion, that may develop right away or even later. Such bleeding in your brain can be fatal. That's why anyone who experiences a brain injury needs to be monitored in the hours afterward and receive emergency care if symptoms worsen.

Risk factors

Factors that may increase your risk of a concussion include:

- Participating in a high risk sport, such as football, hockey, soccer or other contact sport; the risk is further increased if there's a lack of proper safety equipment and supervision
- Being involved in a motor vehicle collision
- Being a soldier involved in combat
- Being a victim of physical abuse
- Falling, especially in young children and older adults
- Having had a previous concussion

Complications

Potential complications of concussion include:

- **Epilepsy.** People who have had a concussion double their risk of developing epilepsy within the first five years after the injury.
- **Cumulative effects of multiple brain injuries.** Evidence exists indicating that people who have had multiple concussive brain injuries over the course of their lives may acquire lasting, and even progressive, cognitive impairment that limits functional ability.

Second impact syndrome. Sometimes, experiencing a second concussion before signs and symptoms of a first concussion have resolved may result in rapid and typically fatal brain swelling. After a concussion, the levels of brain chemicals are altered. It usually takes about a week for these levels to stabilize again. However, the time it takes to recover from a concussion is variable, and it is important for athletes never to return to sports while they're still experiencing signs and symptoms of concussion.

Tumors

Symptoms: Cerebral neoplasms produce subacute and progressive neurologic signs and symptoms. The patient’s symptoms are determined by the size, location and rate of growth of the tumor, as well as the degree of peritumor cerebral edema.

1. Headache from increased intracranial pressure, at times with nausea and vomiting.
2. Focal clinical manifestations (hemiparesis, ataxia, aphasia, visual loss) depend on location and extent of surrounding brain edema. Tumors in relatively "silent" regions of the brain commonly present with changes in personality and behavior.
3. Seizures occur in about one-third of patients with tumors in the supratentorial compartment. Seizures are more likely to accompany slower-growing tumors than highly malignant ones.
4. Hemorrhage into a highly vascular tumor can produce a sudden change in neurological status that can be mistaken for a stroke.

Tumor-Related Brain Edema: Most brain tumors (primary or metastatic) cause edema in the surrounding brain parenchyma. If the edema is considerable or widespread, it can produce a marked increase in intracranial pressure, causing neurologic defects by compressing nearby structures.

Glioblastoma Multiforme (Spongioblastoma Multiforme)
Peak incidence between ages 50 and 60; twice as common in males; most common glioma. Unencapsulated, highly malignant; grows rapidly and infiltrates the brain extensively; may become enormous before diagnosed. Occurs most often in cerebral hemispheres, especially frontal and temporal lobes (rarely in brain stem and cerebellum). Occupies more than one lobe of affected hemisphere; may spread to opposite hemisphere by corpus callosum; may metastasize into cerebrospinal fluid (CSF), producing tumors in distant parts of the central nervous system.

Increased intracranial pressure (ICP), causing nausea, vomiting, headache, papilledema, mental and behavioral changes, altered vital signs (increased systolic pressure, widened pulse pressure, respiratory changes), speech and sensory disturbances. In children, irritability, projectile vomiting.

Astrocytoma
Second most common malignant glioma (30% of all gliomas). Occurs at any age, incidence higher in males. Occurs most often in white matter of cerebral hemispheres; may originate in any part of the central nervous system. Cerebellar astrocytomas usually confined to one hemisphere. Headache, mental activity changes, decreased motor strength and coordination, seizures, scanning speech, altered vital signs.

Oligodendroglioma
Third most common glioma (less then 5%). Occurs in middle adult years, more common in women. Slow growing. Mental and behavioral changes. Decreased visual acuity and other visual disturbances. Increased ICP.

Medulloblastoma
Rare glioma. Incidence highest in children ages 4 to 6. affects males more than females. Frequently metastasized via CSF. Increased ICP.

Ependymoma
Rare glioma. Most common in children and young adults. Locates most often in fourth and lateral ventricles. Similar to oligodendroglioma. Increased ICP and obstructive hydrocephalus, depending on tumor size.

Meningioma
Most common nongliomatous brain tumor, constituting 20% of primary brain tumors. Peak incidence among 50 year olds; rare in children; more common in females than males (ratio 3:2). Arises from the meninges. Common locations include parasagittal area, sphenoidal ridge, anterior part of the base of the skull, cerebellopontine angle, spinal canal. Benign, well-circumscribed, highly vascular tumors that compress underlying brain tissue.

Headache, seizures (two-thirds of patients), vomiting, changes in mental activity, similar to schwannomas.

Schwannoma (Acoustic Neurinoma, Neurilemoma, Cerebellopontine Angle Tumor)
Accounts for approximately 10% of all intracranial tumors. Higher incidence in women. Onset of symptoms between ages 30 and 60. Affects the craniospinal nerve sheath, usually cranial nerve VIII; also, V and VII and, to a lesser extent, VI and X on the same side as the tumor. Benign, but often classified as malignant because of its growth patterns; slow-growing, may be present for years before symptoms occur.

Unilateral hearing loss with or without tinnitus. Stiff neck and suboccipital discomfort. Secondary hydrocephalus. Ataxia and uncoordinated movement of one or both arms due to pressure on brain stem and cerebellum.

Neurofibromatosis Type I (von Recklinghausen Disease)
One of the most common inherited CNS disorders, most common autosomal dominant disorder, most common inherited tumor syndrome and occurs in 1:2,500 live births. Cutaneous lesions: Café-au-lait spots appear early childhood; axillary inguinal freckling, and subcutaneous or cutaneous NF.

Plexiform neurofibroma, optic nerve gliomas, scoliosis (30%), scalloped vertebrae (dural ectasia/lateral meningoceles) > underlying NFs.

Neurofibromatosis Type II
MISME: Multiple intracranial schwannomas, meningiomas, and ependymomas.

Bilateral vestibular schwannomas, multiple extra-axial tumors, schwannomas on cranial nerves and spinal nerve roots, and meningiomas on dural surfaces.

Hereditary syndrome. 50% known family history of NF2; 50% new mutations.1 in 40,000-100,000. Most common kinds of symptoms: hearing loss, vertigo, multiple cranial neuropathies.

Pituitary Adenomas
Microadenomas are less than 10mm in diameter. Will be asymptomatic unless they are hypersecreting tumors.

Functional classification: 1. Non-functioning adenomas 2. Hypersecreting adenomas (prolactin, adrenocorticotrophic hormone (ACTH) or growth hormone)

Hyperprolactinemia: Prolactin levels greater than 100ng/mL almost always indicate a tumor. Hyperprolactinemia in women usually causes amenorrhea or galactorrhea. In men, the earliest symptoms are impotence and loss of libido.

Macroadenomas: Larger tumors compress the adjacent normal pituitary gland and produce hypopituitarism. Extension of the tumor above the sella turcica compress the optic chiasm. This causes progressive visualize loss. Most macroadenomas come to attention because of visual loss or due to headache.

Cerebral Metastases
Metastasis to the brain parenchyma or meninges is a common complication of systemic cancer. Approximately 15-20% of patients dying of cancer will have brain metastasis at the time of autopsy. Bronchogenic carcinoma, breast carcinoma, renal cancer and malignant melanoma are the cancers most likely to metastasize to the brain.The formation of a solid mass lesion is the most common type of cerebral metastasis. Metastases reach the brain by hematogenous spread. The frequency of metastasis in these structures is roughly in proportion to the blood flow to the region (cerebral hemispheres>cerebellum>brainstem). These lesions cause symptoms through increased intracranial pressure, destruction of brain tracts, cerebral edema, or seizures (similar to primary brain tumors). Headaches, intellectual or behavioral changes, focal weakness and unsteadiness are the most common presenting signs.

In about 40% of case of brain metastasis, a solitary lesion will be seen; in the remaining 60%, multiple tumors are seen. MRI with contrast is more sensitive for demonstrating small tumors and may show multiple lesions when only one is seen on contrast-enhanced CT.

White Matter Diseases in Adults
Multiple sclerosis (MS) and variants
Acute disseminated encephalomyelitis
Hurst hemorrhagic leukoencephalitis
Vascular disorders
Ischemic arteriolar disease
Boundary zone ischemia (unilateral carotid disease)
Arteritis (systemic lupus erythematosis , sarcoidosis)
Infectious/immune disorders
Acquired immune deficiency syndrome related disorders
Progressive multifocal leukoencephalopathy
Lyme disease

Vasogenic edema
Traumatic shear injury
Radiation injury/necrosis
Metabolic disorders
    Central pontine myelinolysis
    Marchiafava-Bignami disease
    Adult leukodystrophies

Multiple sclerosis is the most common white matter disease and is related to focal areas of demyelination with reactive gliosis in the white matter of the brain, spinal cord, and the optic nerves. Clinical Presentation: Exacerbations and remissions of multifocal neurologic deficits. Impaired or double vision. Fatigue, weakness, numbness, tingling, and gait disturbances. Loss of sphincter control, blindness, paralysis and dementia. Onset 20-40. More common in persons of Western European lineage who live in temperate zones.

Individuals who migrate in early childhood from a low-risk to a high-risk area have the same risk of developing MS as those in the area they move to. If the same move is made after adolescence, the risk remains low.

- Familial incidence.
- HLA antigens: HLA-A3, HLA-B7, HLA-DR2.
- Cerebrospinal Fluid: Mild lymphocytosis, slightly elevated protein, and oligoclonal immunoglobulin bands of IgG on immunoelectrophoresis.
- Visual, auditory and somatosensory evoked responses.
- Lhermitte's Sign: Electric-like shocks spreading down the body on forceful flexion of the head and neck.

Intracranial Aneurysm

- Saccular aneurysms (berry)
- Tend to occur at arterial bifurcations
- Multiple aneurysms in 20% of adults
- Associated with polycystic kidney disease and coarctation of aorta

Symptoms

- Focal neurological deficit by compression of adjacent structures
- Most are asymptomatic or produce nonspecific symptoms until they rupture
- Subarachnoid hemorrhage

Saccular (berry) Aneurysm

- Most common type
- Secondary to congenital weakness of media
- Usually occurs at major vessel bifurcations
- Occurs at the circle of Willis
- Has a neck or stem
- Has a sac that my be partly filled with a blood clot

Fusiform (spindle-shaped) Aneurysm

- Occurs with atherosclerotic disease
- Characterized by irregular vessel dilation
- Develops on internal carotid or basilar arteries
- Rarely ruptures
- Produces brain and cranial nerve compression or CSF obstruction

Mycotic Aneurysm

- Rare
- Associated with septic emboli that occur secondary to bacterial endocarditis
- Develops when emboli lodge in the arterial lumen, causing arteritis; the arterial wall weakens and dilates

Dissecting Aneurysm

- Caused by arteriosclerosis, head injury, syphilis, or trauma during angiography
- Develops when blood is forced between layers of arterial walls, stripping intima from the underlying muscle layer

Traumatic Aneurysm

- Develops in the carotid system
- Associated with fractures and intimal damage
- May thrombose spontaneously

Giant Aneurysm

- Similar to saccular, but larger - 3cm or more in diameter
- Behaves like a space-occupying lesion, producing cerebral tissue compression and cranial nerve damage
- Associated with hypertension

Subarachnoid Hemorrhage

- Usually from rupture of an aneurysm or AVM
- No specific cause in 20%

Symptoms

- Sudden headache of a severity never experienced previously by patient
- Nausea and vomiting
- Loss or impairment of consciousness

Stroke Belt"
Southeastern USA: North Carolina, South Carolina, Georgia, Alabama, Mississippi, Tennessee, Arkansas and Louisiana. Researchers believe that greater-than-average rates of obesity, cigarette smoking and high blood pressure account for the increased risk of death from cardiovascular disease in the Stroke Belt.

Stroke Symptoms

- Numbness or weakness of the face, arm, or leg, especially on one side of the body
- Confusion, trouble speaking or understanding
- Difficulty seeing in one or both eyes
- Trouble walking, dizziness, loss of balance or coordination
- Severe headache with no known cause

Cerebrovascular Accident
Factors that increase the risk of CVA include history of transient ischemic attacks, atherosclerosis, hypertension, electrocardiogram changes, arrhythmias, rheumatic heart disease, diabetes mellitus, gout, postural hypotension, cardiac or myocardial enlargement, high serum triglyceride levels, lack of exercise, use of oral contraceptives, cigarette smoking, and family history of CVA. The major causes of CVA are thrombosis, embolism, and hemorrhage.

Thrombosis
In middle-aged and elderly, people, among whom there is a higher incidence of atherosclerosis, diabetes, and hypertension, thrombosis is the most common cause of CVA. Obstruction of a blood vessel causes the CVA. Typically, the main site of the obstruction is in extracerebral vessels, but sometimes it's intracerebral.

Thrombosis causes ischemia in brain tissue supplied by the affected vessel as well as congestion and edema. The latter may produce more clinical effects than thrombosis itself, but these symptoms subside with the edema.

Thrombosis may develop while the patient sleeps or shortly after he awakens; it can also occur during surgery or after a myocardial infarction. The risk increases with obesity, smoking, or the use of oral contraceptives. Cocaine-induced ischemic stroke is now being seen in younger patients.

Embolism
The second most common cause of CVA, embolism is an occlusion of a blood vessel caused by a fragmented cloth, a tumor, fat, bacteria, or air. It can occur at any age, especially among patients with a history or rheumatic heart disease, endocarditis, post-traumatic valvular disease, myocardial fibrillation and other cardiac arrhythmias, or following open-heart surgery.

The embolus usually develops rapidly – in 10 to 20 seconds – and without warning. When it reaches the cerebral vasculature, it cuts off circulation by lodging in a narrow portion of an artery, most often the middle cerebral artery, causing necrosis and edema.

Hemorrhage
The third most common cause of CVA is hemorrhage. Like embolism, it may occur suddenly, at any age. Such hemorrhage results from chronic hypertension or aneurysms, which cause sudden rupture of a cerebral artery. The rupture diminishes blood supply to the area served by this artery. In addition, blood accumulates deep within the brain, further compressing neural tissue and causing even greater damage.

Carotid Injury
The CCA bifurcates into the ICA and external carotid artery at approximately the level of the fourth cervical vertebral body near the superior border of the thyroid cartilage. Although the external carotid artery is initially anteromedial to the ICA, and ICA quickly courses medially at approximately the C1 or C2 level before entering the skull base. Unlike the lower cervical vertebrae, the lateral articular processes and pedicles of the first through third cervical vertebrae project more anteriorly, with the distal portion of each cervical ICA lying in close proximity just anterior to these.

The ICA enters the carotid canal as the petrous segment, where it is firmly fixed within the petrous bone. With extension of the neck, the carotid canal is elevated stretching and partially fixing the cervical ICA against the lateral masses of the upper cervical spine. Rotation, which largely occurs at the atlantoaxial joint, forces the contralateral lateral mass of C1 anteriorly, further stretching the ICA. Because of this relationship, the cervical ICA is the typical site of injury associated with hyperextension-rotation of the head and neck.

Type I injuries result from a direct blow to the neck. This is the characteristic mechanism in elderly persons with advanced atherosclerotic disease. Impingement of the ICA between the mandible and the cervical spine with acute hyperflexion would injure the vessel in a similar fashion and may account for some of the injuries seen in victims of motor vehicle accidents.

Type II injuries are due to hyperextension and contralateral rotation of the head and neck and are the characteristic injuries seen in victims of motor vehicle accidents. Damage occurs as the ICA is stretched over the lateral masses of the first and second cervical vertebrae. This mechanism accounts for over 90% of blunt injuries to the ICA and tends to affect young patients more frequently, perhaps due to the protection afforded the elderly by their tortuous vessels and less mobile cervical spine.

Type III injuries result from intraoral trauma and are typically seen in a child who has fallen with a hard object, such as a pencil, in their mouth. Type IV injuries result from associated basilar skull fractures.

Carotid Artery Dissection
The disorder occurs in hypertensive individuals who have no evidence of atherosclerotic vessel disease. Other risk factors include smoking and fibromuscular dysplasia. Dissection may occur with hyperextension and lateral flexion of the neck as the artery is stretched over the transverse processes of the upper cervical vertebrae. Focal unilateral headache is the most common symptom in association with dissection. The headache is steady, non-throbbing, of variable intensity, and is located in the frontal,

auricular, or periorbital area. Neurologic manifestations may include stroke (resulting in contralateral hemiparesis, paresthesias, aphasia, ipsilateral blindness, or abducens paralysis) or oculosympathetic palsy with ptosis and miosis without anhidrosis. Focal neurologic deficits may follow the onset of headache or neck pain within minutes of hours. Bruits may be heard over the carotid.

Vertebral Artery Dissection
Vertebral artery dissection occurs most commonly in middle-aged women. People with hypertension or fibromuscular dysplasia are at greater risk of dissection. Pain in the occiput or posterior neck is the presenting symptom in 80% of patients, preceding ischemic symptoms by minutes to 30 days. Most patients present with a completed stroke, with a minority presenting with transient ischemic attacks. The lateral medullary syndrome (pain, numbness, ipsilateral face [trigeminal], ataxia, vertigo, nystagmus, Horner's syndrome [descending sympathetic tract], dysphagia, numbness of ipsilateral appendages) is the most common neurologic manifestation. Severe cases may have basilar artery involvement with associated quadriparesis, dysphagia, diplopia, with preserved sensation.

Symptoms of TIA
Carotid Territory – paresthesia/weakness of hand, arm, and face; aphasia (dominant hemisphere); dysarthria; unilateral neglect.

Lacunar – hemibody sensory loss of paresthesia; pure motor hemiparesis.

Vertebrobasilar – dysarthria; vertigo, ataxia; diplopia; visual field loss; perioral paresthesias; acute confusional state; profound general weakness.

Transient Ischemic Attack
TIA is a sudden or rapid onset of neurological deficit caused by cerebral ischemia. It may last for a few minutes or up to 24 hours and clears without residual signs. Risk factors: hypertension, smoking, obesity, hyperlipidemias, advanced age.

Etiology
1. Cardiac emboli: atrial fibrillation, mitral valve disease, prosthetic heart valves, bacterial and marantic endocarditis, intracardiac defects with paradoxical embolism (patent foramen ovale, atrial septal defect)
2. Carotid or vertebral artery disease: arteriosclerosis, fibromuscular hyperplasia, traumatic and spontaneous carotid and vertebrobasilar artery dissection

Characteristics of Carotid Artery Syndrome
- Ipsilateral monocular vision loss (amaurosis fugax); the patient often feels as if "a shade" has come down over one eye
- Episodic contralateral arm, leg, and face paresis and paresthesias
- Slurred speech, transient aphasia
- Ipsilateral headache of vascular type
- Carotid bruit may be present over the carotid bifurcation
- Microemboli, hemorrhages, and exudates may be noted in the ipsilateral retina

Characteristics of Vertebrobasilar Artery Syndrome

- Binocular visual disturbances (blurred vision, diplopia, total blindness)
- Vertigo, nausea, vomiting, tinnitus
- Sudden loss of postural tone of all four extremities (drop attacks) with no loss of consciousness
- Slurred speech, ataxia, numbness around lips or face

Vertebrobasilar Injuries

Mechanisms of vertebral artery injury: cervical hyperextension, excessive contralateral rotation, and hyperextension and rotation.

Familial history of stroke or cardiovascular disease, hypertension, smoking, cervical spondylosis/arthrosis, bleeding disorders, medication, and/or anatomical anomaly/pathology.

Rotation of C1 on C2 between 30 and 45º causes the vertebral artery at the atlantoaxial junction to become compressed on the opposite side of head rotation, subsequently reducing blood flow to the basilar artery. In the normal patient, this diminution of blood flow caused by positional change of the cervical spine will not cause any neurological symptoms, such as dizziness, nausea, tinnitus, faintness, or nystagmus. This lack of symptoms is a result of the normal flow of collateral circulation by the opposite vertebral artery, common carotid arteries, and a communicating cerebral arterial circle (Circle of Willis).

Seven areas of possible compression: 1) between C1-2 transverse processes, where the vertebral arteries are relatively fixed at the C1 and C2 transverse foramina; 2) C2-3 at the level of the superior articular facet of C3 on the ipsilateral side to head rotation; 3) the C1 transverse process and the internal carotid artery; 4) the atlanto-occipital aperture by the posterior arch of atlas and the rim of foramen magnum, or anteriorly by folding of the atlanto-occipital joint capsule and posteriorly by the atlanto-occipital membrane; 5) C4-5 or C5-6 levels because of arthrosis of the joints of von Luschka with compression on the ipsilateral side to head rotation; 6) at the transverse foramina of the atlas or axis between the obliquus capitis inferior and intertransversarii during rotatory movements; 7) before entering the C6 transverse process by the longus colli muscle or by tissue communicating between the longus colli and scalenus anterior muscles.

Vertebral artery pathological alterations: intimal disruption, subintimal hematoma, dissection, pseudoaneurysm, and thromboembolism.

Wallenberg's Syndrome: ipsilateral loss of cranial nerves V, IX, X, and XI cerebellar ataxia, Horner's syndrome, and contralateral loss of pain and temperature sensation. Sudden death, quadriplegia, and the "locked-in" syndrome (quadriplegia with loss of all lower cranial nerves).

Risk factors: age, hypertension, hyperlipidemia, family history of stroke or heart attacks, diabetes, smoking, heart and peripheral vascular disease, young adult females on birth control pills, cervical spondylosis, and cervical spine injury (hyperextension injury).

Clinical evaluation: blood pressure (both arms), palpate radial pulses (normal, feeble or absent), palpate carotid pulses, auscultate carotid arteries (bruit, hissing or squirting sound), and auscultate subclavian arteries. If pulsations or bruits are present at the carotid or subclavian arteries, do not perform the functional maneuver. A difference of 10 mm Hg between the two systolic blood pressures and a feeble or absent radial pulse is suggestive of subclavian artery stenosis.

Atherosclerosis
Pathologic degenerative process resulting from deposition of plasma lipids in arterial walls. Location: Internal carotid and basilar arteries most common sites in head and neck. Plaque surface irregularity associated with increased risk of stroke at all degrees of stenosis. Endarterectomy if symptomatic carotid stenosis is ≥ 70%.

	Lower Motor Neuron Syndrome	Upper Motor Neuron Syndrome
Structures involved	Anterior horn cell, root, nerve, neuromuscular junction, muscle	Cerebrum, brain stem, spinal cord
Muscles affected	Individual muscles	Groups of muscles
Wasting	Present, often marked	Absent
Fasciculations	Present	Absent
Tone	Flaccidity	Spasticity
Tendon reflexes	Decreased or absent	Hyperactive
Clonus	Absent	Present
Plantar responses	Flexor	Extensor (Babinski sign)
Superficial abdominal and cremasteric reflexes	Present	Absent
Electromyography (EMG)	Abnormal	Normal

Metastatic brain tumors are 10 times more common than primary brain tumors.

Primary Brain Tumors

Fifty percent of primary tumors are of glial origin. Most common subtype is glioblastoma (50% of all gliomas, 25% of all primary brain tumors). The most common benign primary brain tumor is meningioma (30% of all primary brain tumors). Pituitary adenomas are the third most common brain tumor. Primary brain tumors have a slight male predominance. Peaks of incidence in childhood and older adulthood.

MRI
High-grade gliomas and metastases appear as contrast-enhancing intra-axial mass legions surrounded by edema. Low-grade gliomas are typically nonenhancing. Low-grade gliomas best appreciated with T2/FLAIR.
MRI FLAIR suppresses CSF T2 signal for better lesion identification.

Neoplasms of Nerve Roots, Dura and Spinal Cord. Extradural. Intradural extramedullary. Intramedullary.

Incidence of Intraspinal Neoplasms
Schwannomas 30%. Meningiomas 26%. Gliomas 23%. Sarcomas 11%. Hemangiomas 6%. Other 4%.

Glioblastoma Multiforme (Spongioblastoma Multiforme) Peak incidence at 50 to 60 years: twice as common in males; most common glioma (accounts for 60% of all gliomas). Unencapsulated, highly malignant; grows rapidly and infiltrates the brain extensively; may become enormous before diagnosed. Usually occurs in cerebral hemispheres, especially frontal and temporal lobes (rarely in brain stem and cerebellum). Occupies more than one lobe of affected hemisphere; may spread to opposite hemisphere by corpus callosum; may metastasize into cerebrospinal fluid (CSF), producing tumors in distant parts of the nervous systemerebrospinal fluid (CSF), producing tumors in distant parts of the nervous system.

Brain Tumors-Signs and Symptoms-Localizing Midline: headache (bifrontal or bioccipital); worse in the morning; intensified by coughing, straining, or sudden head movements. Temporal lobe: psychomotor seizures. Central region: focal seizures. Optic and oculomotor nerves; visual defects. Frontal lobe: abnormal reflexes, motor responses.

Brain Tumors-Signs and Symptoms-General Increased ICP (nausea, vomiting, headache, papilledema). Mental and behavioral changes. Altered vital signs (increased systolic pressure, widened pulse pressure, respiratory changes). Speech and sensory disturbances. In children, irritability, projectile vomiting

Low-grade tumors are more epileptogenic than high-grade tumors and metastases.

Cysts Associated with Spinal Cord Tumors
Tumoral cyst: Degeneration, necrosis, and liquefaction within the neoplasm. Contains a mixture of differing elements such as protein, old hemorrhage, and necrotic tumoral tissue. Cephalic or caudal cysts: Occur above and/or below the tumor. Contain either hemorrhagic or xanthochromic fluid. Reactive dilatation of the central canal: Most likely related to partial obstruction of the central canal.

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Oligodendroglioma Third most common glioma (accounts for less than 5% of all gliomas). Occurs in middle adult years; more common in women. Slow-growing.

Medulloblastoma Rare glioma. Incidence highest in children ages 4 to 6. Affects males more commonly than females. Commonly metastasizes via CSF.

Schwannoma Neurilemoma, neurinoma, neuroma, vestibular schwannoma . Benign encapsulated nerve sheath tumor composed of differentiated neoplastic schwann cells " looks like "ice cream on cone". 98% of intracerebral schwannomas arise from cranial nerves (predominately sensory). 90% arise from CN 8 (vestibular portion). 10% other (5% CN 5 & 7).

Schwannoma (Acoustic Neurinoma, Neurilemoma, Cerebellopontine Angle Tumor) Accounts for about 10% of all intracranial tumors. Higher incidence in women. Onset of symptoms between ages 30 and 60. Affects the craniospinal nerve sheath, usually cranial nerve (CN) VIII; also, CN V and VII and, to a lesser extent, CN VI and X on the same side as the tumor. Benign, but commonly classified as malignant because of its growth patterns; slow-growing – may be present for years before symptoms occur.

Schwannoma-Signs and Symptoms-General Unilateral hearing loss with or without tinnitus. Stiff neck and suboccipital discomfort. Secondary hydrocephalus. Ataxia and uncoordinated movements of one or both arms due to pressure on brain stem and cerebellum. **Schwannoma-Signs and Symptoms-Localizing** CN V: early-facial hypoesthesia or paresthesia on side of hearing loss; unilateral loss of corneal reflex. CN VI: diplopia or double vision. CN VII: paresis progressing to paralysis (Bell's palsy). CN X: weakness of palate, tongue, and nerve muscles on same side as tumor.

Meningioma Most common nongliomatous brain tumor (15% of primary brain tumors). Peak incidence among 50 year-olds; rare in children; more common in females (ratio 3:2). Arises from the meninges. Common locations include parasagittal area, sphenoidal ridge, anterior part of the base of the skull, cerebellopontine angle, spinal canal. Benign, well-circumscribed, highly vascular tumors that compress underlying brain tissue by invading overlying skull.

Meningioma-Signs and Symptoms-General Headache. Seizures (in two thirds of patients). Vomiting. Changes in mental activity.

Meningioma-Signs and Symptoms-Localizing Skull changes (bony bulge) over tumor. Sphenoidal ridge, indenting optic nerve: unilateral visual changes and papilledema. Prefrontal parasagittal: personality and behavioral changes. Motor cortex contralateral motor changes. Anterior fossa compressing both optic nerves and frontal lobes: headaches and bilateral vision loss. Pressure on cranial nerves causing varying symptoms.

Meningioma
Dural-based enhancing mass w/cortical buckling & trapped CSF clefts/cortical vessels. Supratentorial (90%): Parasagittal/convexity (45%), sphenoid ridge (15-205), olfactory groove (5-10%), parasellar (5-10%). Infratentorial (8-10%): CPA most common. Peritumoral hypodense vasogenic edema (60%) .> 95% enhance homogeneously & intensely. Dural "tail" (35-80% of cases): non-specific.

Parenchymal Metastases
Discrete parenchymal mass(es) at gray-white interface. Discrete, focal mass(es) at arterial border zones
80% hemispheres. Size: Varies from microscopic to several cm. Most mets are discrete, spherical. 50% of metastases are solitary. 20% two metastases, 30% three or more.

Pituitary Adenoma
Microadenoma < 10mm in diameter. Macroadenoma > 10mm. Sellar mass without separate identifiable pituitary gland. Intra- or combined intra-suprasellar. Most common suprasellar mass in adults. “Figure-of-eight” or “snowman”.

Pyogenic spondylodiscitis. Ill-defined hypointense vertebral marrow on T1W1 with loss of endplate definition on both sides of the disc. Lumbar (48%)>thoracic (35%)>cervical spine (6%).

Multiple Sclerosis
MS is a progressive disease caused by demyelination of the white matter of the brain and spinal cord. Sporadic patches of demyelination throughout the central nervous system induce widely disseminated and varied neurologic dysfunction. Characterized by exacerbations and remissions. Prognosis varies; MS may progress rapidly, disabling some patients by early adulthood or causing death within months of onset. However, 70% of patients lead active, productive lives with prolonged remissions.

MS - Causes and Incidences Current theories suggest a slow-acting or latent viral infection and an autoimmune response. MS usually begins between the ages of 20 and 40. It affects more women than men. A family history of MS and living in a geographical area with higher incidence of MS (northern Europe, northern United States, southern Australia, and New Zealand) increase the risk.

MS – Signs and Symptoms May be transient or they may last for hours or weeks. They may wax and wane with no predictable pattern, vary from day to day, and be bizarre and difficult for the patient to describe. Visual disturbances and sensory impairment, such as numbness and tingling sensations (paresthesia) are the first signs that something may be wrong. Occular disturbances – optic neuritis, diplopia, ophthalmoplegia, blurred vision, and nystagmus. Muscle dysfunction – weakness, paralysis ranging from monoplegia to quadriplegia, spasticity, hyperreflexia, intention tremor, and gait ataxia. Urinary disturbances – incontinence, frequency, urgency, and frequent infections. Emotional lability – characteristic mood swings, irritability, euphoria, and depression. Most common neurologic disease among young adults. Incidence is highest from ages 20 to 40, but can start in childhood or after age 50. Female-to-male ratio: 7:3. Prevalence decreases with proximity to equator. U.S. prevalence: 400,000+. In the U.S. there are 8,500-10,000 new cases per year.

MS Genetic Risk. General population: 0.1%. If one parent or first-degree relative is affected: 4%. If both parents are affected: 20%.

MS – Signs and Symptoms Constellation depends on location of lesion(s) within brain, spinal cord, and optic nerves. Initially, attacks of inflammation and CNS

dysfunction are usually followed by full recovery, but over time, deficits may persist. Attacks typically worsen over several days, plateau, and then improve over days to weeks. Most common symptoms include:Visual/oculomotor disturbances (49%). Leg paresis/leg paresthesias (42%). Cerebellar ataxia (24%). Cognitive impairment (4%). Cerebellar ataxia (24%)

MS - Other Symptoms Include: Lhermitte's phenomenon: Electrical paresthesias induced by neck flexion. Uthoff's phenomenon: Worsening symptoms/signs with increased body temperature (showering, exercising). Neuropathic pain. Fatigue.

MS – Pathology/Pathophysiology Historically considered a disease of white matter, but recent data suggest additional neurodegeneration and primary involvement of gray matter. Epstein-Barr virus (EBV) and human herpesvirus 6 (HSV-1) are most consistently and recently implicated. Other possibilities include measles, rubella, mumps, coronavirus, parainfluenza, herpes simplex virus type 1 (HSV-1), vaccinia, and human T-lymphotropic virus type 1 (HTLV-1). The pathologic hallmarks of MS are demyelination and inflammation, predominantly perivenular. Severe or advanced disease also involves axonal disruption and cortical atrophy/neurodegeneration.

Classification by Disease Course Benign MS: 5% of all patients. Relapsing-remitting (RRMS): 85% at initial diagnosis. Primary progressive (PPMS): 10-20% at initial diagnosis. Secondary progressive (SPMS): 60% of RRMS becomes SPMS after 15 years.

Favorable Prognostic Indicators Younger age of onset. Female sex. Monosymptomatic onset. Sensory rather than motor symptoms at onset. Few T2/FLAIR lesions on original MRI. Long interval between first and second attacks. Low attack frequency in the first 2 years. Full recovery of function after the first attack.

MRI Findings in MS Dawson's fingers: T2 hyperintensities arranged perpendicular to the plane of lateral ventricles. Reflect demyelination and perivenular inflammation. T2/FLAIR hyperintensities: Reflect total disease burden, including reversible and irreversible pathology. T1 gadolinium enhancement: Inflammation, blood-brain barrier disruption, and recent disease activity (<8 weeks) with new lesion formation. T1 hypointensities ("black holes"): Reflect severe tissue pathology, axonal loss, and clinical disability. Global and focal cerebral atrophy measures in brain and spinal cord: Correlate with axonal loss, neuronal loss, physical and cognitive impairment.

Benign MS: No or minimal neurologic disability after 10-15 years: 5% of all patient. Malignant MS: Neurologic disability requiring ambulation assistance after ≤ 5 years – 5-7% of all patients.

MS - T2-Weighted MRI
Inflammation. High sensitivity to detect new lesions and changes in lesion size over time. Low pathologic specificity. Poor correlation with disability. Cannot distinguish between edema, gliosis, demyelination and axonal loss.

MS - T1-Weighted MRI
Tissue destruction. More pathologically specific than conventional T2-weighted MRI.

MRI - Acute Hypointense Lesions

Tissue destruction. Edema associated with inflammation (acute hypointense lesions).
Tissue destruction with demyelination and axonal loss (chronic hypointense lesions).

MRI - Gadolinium-Enhanced T1 Weighting

Blood-brain barrier integrity. Identifies early inflammatory phase of lesion development with active phagocytosis by macrophages. Measures inflammatory activity and demyelination. Permits active and inactive lesions to be differentiated.

MS

Persistent hypointense T1-weighted images correlate more closely with disability than do T2-weighted images and are known as “black holes”.

MS

The appearance of three or more white matter lesions on a T2-weighted MR scan is a sensitive predictor (>80%) of the development of clinically definite MS within 7 to 10 years.

MS

Also highly predictive of subsequent development of clinically definite MS are the appearance of two or more gadolinium-enhanced lesions at baseline and the presence of either new T2 lesions or new gadolinium-enhanced lesions three months or more after a clinically isolated demyelinating event.

Characteristic MRI Features of MS Lesions

Immediate proximity to the ventricles, especially in a confluent, poorly demarcated, “lumpy-bumpy” pattern; vertical (perpendicular) orientation to the ventricles is also common. Lesions > 6mm in diameter. Infratentorial or corpus callosum lesions.

Differential Diagnosis of MRI White Matter Changes in MS

Cerebrovascular disease. Migrainous ischemia. Vasculitis. Lacunes
Binswanger’s disease. Thromboembolic infarcts. Moyamoya disease.
Acute disseminated encephalomyelitis.Progressive multifocal leukoencephalopathy. Inherited white matter diseases. Effects of radiation therapy. Metastatic neoplasm. Primary CNS lymphoma. Lyme disease. HTLV-1 infection. “Normal”, especially if elderly or hypertensive.

Clinically Isolated Syndrome (CIS) MS often presents for the first time with a clinically isolated syndrome (CIS) such as optic neuritis (ON), an acute brain stem syndrome, or transverse myelitis (TM).

Optic Neuritis Acute-subacute inflammation of the optic nerve with visual loss. Acute/subacute unilateral decrease or loss of vision. Central vision most affected. Color vision impaired. Orbital pain with eye movement common.

Acute Transverse Myelitis
Central cord lesion more than two vertebral segments in length with eccentric enhancement. Thoracic more common. High signal intensity. Variable post-gadolinium enhancement. Up to 40-50% of cases not demonstrated by MRI. ATM can mimic cord neoplasm.

Transverse Myelitis Acute/subacute inflammation of the spinal cord. Usually limited to ≤ 3 vertebral segments and occupies less than two-thirds of cross-sectional cord diameter.
Symptoms Ascending numbness and paresthesias in the legs. Sensory symptoms involving the trunk and the perineum. Difficulty with or loss of bladder and/or bowel control. Leg weakness. Sensory and/or motor symptoms involving the arms (< 20%). Back pain.

Neuromyelitis Optica (Devic's Disease) Severe acute myelitis (usually cervical) and bilateral optic neuritis. Usually monophasic. Common in Africa and Asia, rarer in North America. Usually spans > 3 spinal segments; often involves swelling of the spinal cord. Pathology: Extensive demyelination, cavitating necrosis, acute axonal injury, loss of oligodendrocytes.

Acute Disseminated Encephalomyelitis (ADEM) Monophasic, acute, demyelinating inflammatory illness, typically following upper respiratory infection (50-75%) or vaccination. More common in children. First symptoms 7-14 days post infection, most hospitalized within a week.

ADEM
Following nonspecific upper respiratory tract infection, often viral. After specific viral illness: Epstein-Barr, influenza A, mumps, coronavirus. Especially after exanthematous diseases of childhood (chickenpox, measles). After vaccination: Diphtheria, influenza, rabies, smallpox, tetanus, typhoid

Signs and Symptoms (ADEM) Children > adults: Prolonged fever, headache, imbalance/gait instability, dysphagia/dysarthria, diplopia. Adults > children: Limb paresthesias and weakness. Confusion/disorientation, altered alertness. Gait ataxia, dysmetria, dysarthria, brain stem signs, sensory disturbances, pyramidal weakness. Suspect ADEM when: Close temporal relation to infection or vaccination. MRI shows > 50% involvement of white matter; may also involve deep gray.

ADEM - Acute Disseminated Encephalomyelitis
Multifocal WM/basal ganglia lesions 10-14 days following infection/vaccination. May involve both brain and spinal cord; predominantly WM but also gray matter. Multifocal punctate to large flocculent FLAIR hyperintensities. May appear identical to MS; repeat MR necessary to distinguish with certainty.

Cerebral Venous Thrombosis Thrombosis of dural venous sinuses → elevated intracranial pressure,

Symptoms Sudden-onset headache ("worst headache in my life"). Meningismus. Fever. Nausea/vomiting. Seizures. Coma. Patients complain of new-onset headache within the preceding 1-7 days in about 30% of cases, likely due to sentinel leak prior to major aneurysm rupture.

Intracerebral Hemorrhage Occurs most commonly in putamen (50%) followed by cortex, thalamus, cerebellum, pons, respectively. Blood pressure is the number one modifiable risk factor.

Arteriovenous Malformation (AVM) Tangle of abnormal, tortuous arteriovenous fistulas that lack an intervening capillary bed and are sparated by a nidus of brain tissue; most commonly supratentorial. Present with headache, seizures, or hemorrhage; 2% annual hemorrhage rate, 10-20% recurrence rate in first year after hemorrhage.

Cavernous Hemangioma Cluster of thin-walled veins without significant arterial feeders or intervening brain tissue; infratentorial in 50%, multiple in 10%, and familial (autosomal dominant) in 5%; hemorrhage rate 1-2%/year.

Underlying Tumor Most common tumors that bleed are breast, lung, thyroid, renal cell, melanoma and choriocarcinoma.

Cerebral Venous Thrombosis Thrombosis of dural venous sinuses → elevated intracranial pressure, cerebral edema, ischemia, and hemorrhage. Symptoms and findings are caused by impedance of venous drainage of blood and CSF. Most commonly involves the superior sagittal sinus. Should be suspected in young patients (30s and 40s) with focal neurological deficits and signs of elevated intracranial pressure.

Symptoms Headache: Worse while dependent or with straining. Nausea/vomiting. Meningismus with or without fever. Focal neurological deficits. Visual blurring or blindness.

Subarachnoid Hemorrhage Presence of blood in the CSF-filled space underlying the arachnoid layer of the meninges related to trauma or rupture of a vascular structure. The most common cause of subarachnoid hemorrhage (SAH) is head trauma. In the absence of trauma, rupture of a cerebral aneurysm or arteriovenous malformation should be suspected. The most common locations for aneurysms include posterior communicating artery, anterior communicating artery, middle cerebral artery and basilar artery (in order).

Symptoms Sudden-onset headache (“worst headache in my life”). Meningismus. Fever. Nausea/vomiting. Seizures. Coma. Patients complain of new-onset headache within the preceding 1-7 days in about 30% of cases, likely due to sentinel leak prior to major aneurysm rupture.

Vertebral or Carotid Dissection Intimal tear of the vertebral or carotid artery at the skull base resulting in stenosis or occlusion of the parent or feeder vessels and potential distal embolization, typically associated with neck pain with a “tearing” quality.

Causes of Stroke

Atrial fibrillation: Turbulent/static blood clots within dilated left atrium or atrial appendage → embolic stroke (MCA territory most common).
Vertebral or Carotid Dissection Intimal tear of the vertebral or carotid artery at the skull base resulting in stenosis or occlusion of the parent or feeder vessels and potential distal embolization, typically associated with neck pain with a "tearing" quality.
Patent Foramen Ovale Congenital anomaly resulting in paradoxical embolization of venous thrombi via a right → left shunt due to failure of closure of the fetal foramen ovale.
Carotid Stenosis Atherosclerosis of the carotid artery just distal to the carotid bifurcation, caused by turbulent blood flow and resulting in distal emboli, typically in the MCA territory.
Intracranial Atherosclerosis Focal atherosclerosis of the large intracranial vessels of the circle of Willis →recurrent strokes of TIA's in the same vascular territory or blood pressure-dependent focal neurological deficits. More common in African-Americans and Asians, whereas extracranial atherosclerosis is more common in Caucasians.
Dilated Cardiomyopathy Turbulent blood flow within dilated ventricle →embolic stroke, typically in the MCA distribution.
Watershed Infarcts Ischemic stroke involving the watershed region between two vascular territories due to focal or generalized reduction in the perfusion pressure. Most commonly occurs between the MCA and ACA territories →bilateral infarcts along the medical cortical and subcortical regions →bilateral proximal arm/leg and trunk weakness (man-in-barrel syndrome).

Traumatic Brain Injury (TBI) Etiology. < 75 years of age: Most commonly associated with motor vehicle collision. > 75 years of age: Most commonly secondary to falls. Alcohol related: 50%. Violence related: 20%.

Epidural Hematoma Hemorrhage between the dura and skull periosteum, typically due to tearing of the middle meningeal artery associated with temporal bone fracture. Head CT: Convex (lens shaped) hyperdensity that does not cross skull suture lines.

Subdural Hematoma Tearing of bridging veins (venous sinuses) due to shearing forces with trauma associated with rotational acceleration/deceleration (falls and assaults). May occur without a blow to the head (i.e. whiplash injury) in older patients due to movement of an atrophic brain with respect to the dura.

Cerebral Contusions Parynchymal bruising of brain due to blunt head trauma. Coup/contre-coup injuries: Blows to the back of the head result in contusion of the orbital frontal and anterior temporal lobes as the brain strikes the rigid orbital plate and sphenoid wing, respectively.

Diffuse Axonal Injury (DAI) Acceleration/deceleration injury →shearing/tearing of axons, typically seen with rotational/angular as opposed to linear forces. Most commonly occurs at gray-white junction and large white matter tracts such as the corpus callosum and middle of cerebral peduncles.

Giant Cell Tumor
Most common tumor of sacrum in young adults. 10-15% have ABC component. Lytic expansile lesion.

ABC
10-30% occur in spine/sacrum. Arise in neural arch. 75-90% extend into VB. Absent pedicle sign. Fluid- levels caused by hemorrhage, blood product sedimentation.

Arachnoid Cyst
Spinal Meningeal Cyst. Intraspinal extramedullary loculated cerebral spinal fluid collection. Nonenhancing extramedullary loculated CSF intensity collection displacing cord or nerve roots. Extradural or intradural extramedullary. Suggested by mass effect on cord and nerve roots.

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Arachnoid Cyst
Spinal Meningeal Cyst. Extradural: Posterior or posterolateral lower thoracic spine. Intradural: Dorsal mid thoracic spine. Anterior location uncommon. Cyst wall may be imperceptible.

Neurofibromatosis Type 2
Bilateral vestibular schwannomas. Multiple extra-axial tumors. Schwannomas on cranial nerves and spinal nerve roots. Meningiomas on dural surfaces. Intra-axial tumors. Ependymomas in spinal cord and brainstem.

Neurofibromatosis Type 1 - Von Recklinghausen Disease
Neurofibromatosis type 1. Focal areas of signal intensity in white matter & deep gray matter. Plexiform neurofibroma and optic nerve gliomas.

NF1
Neurocutaneous disorder, phakomatosis. Inherited tumor disorder characterized by diffuse neurofibromas, intracranial hamartomas, benign & malignant tumors. Most common autosomal dominant disorder. Most common inherited tumor syndrome. 1:2, 500 live births. Cutaneous lesions: Café-au-lait spots appear early childhood; axillary or inguinal freckling childhood and adolescence; subcutaneous or cutaneous NF when older.

Epidural Lipomatosis
Excessive accumulation of intraspinal fat causing cord compression and neurologic deficits. Abundant epidural fat in mid-thoracic and distal lumbar spinal canal compressing thecal sac. Thoracic spine: 58-61%. Lumbar spine: 39-42%. Epidural fat ≥ 7mm thick in thoracic spine. Steroid use: 75% of reported cases.