

2011 Imaging Criteria

Magnetic Resonance Imaging (MRI), Brain (Pediatric)^(1, 2, 3)

ICD-9-CM: 88.91

CPT: 70551, 70552, 70553

I/O Setting: Outpatient

INDICATION(S)

- 100 Acute onset persistent neurologic Sx/findings ♦
- 200 New transient neurologic Sx/findings ♦
- 300 Headache
- 400 Seizure
- 500 Head trauma ♦
- 600 Suspected nonaccidental head trauma
- 700 Suspected CNS infection
- 800 Follow-up of intracranial abscess
- 900 Follow-up of primary brain tumor
- 1000 Single brain tumor by CT
- 1100 CNS evaluation for brain metastases
- 1200 Follow-up of AVM
- 1300 Post intracranial procedure/craniotomy/craniectomy
- 1400 Suspected CNS involvement with systemic disease
- 1500 Multiple sclerosis (MS)
- 1600 Suspected neurodegenerative disorder
- 1700 Suspected hydrocephalus
- 1800 Nonacute onset mental status changes
- 1900 Macrocephaly
- 2000 Microcephaly

- 100 Acute onset persistent neurologic Sx/findings **[One]** ♦⁽⁴⁾
 - 110 Sensory deficit⁽⁵⁾
 - 120 Motor deficit⁽⁶⁾
 - 130 Language deficit⁽⁷⁾
 - 140 Cognitive dysfunction of unknown etiology^(8, 9)
 - 150 Visual impairment⁽¹⁰⁾
 - 160 Altered level of consciousness⁽¹¹⁾

- 200 New transient neurologic Sx/findings **[One]** ♦
 - 210 Sensory deficit⁽⁵⁾
 - 220 Motor deficit^(6, 12)

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- 230 Language deficit⁽⁷⁾
- 240 Visual impairment⁽¹⁰⁾
- 250 Memory loss by Hx/PE⁽¹³⁾

- 300 Headache [**One**]⁽¹⁴⁾
 - 310 Papilledema by PE^(15, 16, 17)
 - 320 New headache [**One**]⁽¹⁸⁾
 - 321 Focal neurologic finding by PE⁽¹⁹⁾
 - 322 Headache with syncope by Hx⁽²⁰⁾
 - 323 Mental status changes by Hx/PE⁽²¹⁾
 - 324 Headache onset with exertion/Valsalva maneuver by Hx
 - 325 Headache causes awakening from sleep by Hx⁽²²⁾
 - 326 Headache with nocturnal vomiting by Hx⁽²³⁾
 - 330 Chronic headache [**One**]⁽²⁴⁾
 - 331 Focal neurologic finding by PE⁽¹⁹⁾
 - 332 Headache with syncope by Hx⁽²⁰⁾
 - 333 Mental status changes by Hx/PE⁽²¹⁾
 - 334 Worsening of previously stable chronic headache by Hx⁽²⁵⁾

- 400 Seizure [**One**]⁽²⁶⁾
 - 410 Seizure with focal neurologic finding by PE^(27, 28)
 - 420 New onset seizure and patient afebrile ♦^(29, 30)
 - 430 Refractory to Rx [**All**]^(31, 32, 33)
 - 431 Increased seizure activity with therapeutic blood levels of anticonvulsant
 - 432 ≥ 12 wks since initiation of anticonvulsant Rx⁽³⁴⁾
 - 433 No concurrent seizure-provoking medication⁽³⁵⁾

- 500 Head trauma [**Both**] ♦⁽³⁶⁾
 - 510 Findings [**One**]⁽³⁷⁾
 - 511 Age ≤ 6 mos⁽³⁷⁾
 - 512 Sx/findings [**One**]
 - 1 Retrograde/anterograde amnesia^(38, 39)
 - 2 LOC by Hx/PE
 - 3 Mental status changes by Hx/PE⁽²¹⁾
 - 4 Irritability/behavioral changes by Hx/PE
 - 5 Vomiting⁽⁴⁰⁾
 - 6 Bulging fontanel by PE
 - 7 Focal neurologic finding by PE⁽¹⁹⁾
 - 8 Headache by Hx
 - 9 Seizure by Hx/PE
 - 10 Coagulopathy by Hx

- 11 Skull fracture by PE/x-ray⁽⁴¹⁾
- 520 CT not feasible/nondiagnostic for etiology of Sx/findings⁽⁴²⁾
- 600 Suspected nonaccidental head trauma **[Both]**⁽⁴³⁾
 - 610 Sx/findings **[One]**
 - 611 Irritability/behavioral changes by Hx/PE
 - 612 Seizure by Hx/PE
 - 613 Retinal hemorrhage⁽⁴⁴⁾
 - 614 Cutaneous bruising/swelling
 - 615 Multiple/bilateral skull fracture by PE/x-ray⁽⁴⁵⁾
 - 616 Vague/absent Hx of head trauma with findings by PE⁽⁴⁶⁾
 - 620 CT nondiagnostic for etiology of Sx/findings⁽⁴⁷⁾
- 700 Suspected CNS infection **[One]**^(48, 49)
 - 710 New/worsening CNS Sx/findings **[One]** ♦⁽⁵⁰⁾
 - 711 Focal neurologic finding by PE⁽¹⁹⁾
 - 712 Headache by Hx
 - 713 Meningismus^(51, 52)
 - 714 Mental status changes by Hx/PE⁽²¹⁾
 - 715 Seizure by Hx/PE
 - 720 Follow-up assessment⁽⁵³⁾
- 800 Follow-up of intracranial abscess **[One]**^(54, 55, 56)
 - 810 New/worsening CNS Sx/findings **[One]** ♦⁽¹⁹⁾
 - 811 Focal neurologic finding by PE⁽¹⁹⁾
 - 812 Vomiting
 - 813 Headache by Hx
 - 814 Mental status changes by Hx/PE⁽²¹⁾
 - 815 Seizure by Hx/PE
 - 820 New onset temperature > 100.4 F(38.0 C)⁽⁵⁷⁾
 - 830 Continued temperature > 100.4 F(38.0 C) after 1 week IV Abx Rx⁽⁵⁷⁾
 - 840 Follow-up assessment during Rx⁽⁵⁸⁾
 - 850 Follow-up assessment after Rx completed
- 900 Follow-up of primary brain tumor **[One]**^(59*RIN)
 - 910 New/worsening CNS Sx/findings ♦^(60, 61)
 - 920 Periodic assessment
- 1000 Single brain tumor by CT^(61, 62)

- 1100 CNS evaluation for brain metastases **[One]**
 - 1110 Baseline scan as part of staging **[One]**
 - 1111 Sarcoma
 - 1112 Melanoma
 - 1120 Baseline scan positive **[One]**
 - 1121 Periodic assessment during chemotherapy/radiation Rx⁽⁶³⁾
 - 1122 Restaging after chemotherapy/radiation Rx completed⁽⁶⁴⁾
 - 1130 New/worsening CNS Sx/findings **[One]** ♦
 - 1131 Known cancer elsewhere
 - 1132 Known brain metastasis by prior CT/MRI

- 1200 Follow-up of AVM^(65, 66)

- 1300 Post intracranial procedure/craniotomy/craniectomy **[One]**⁽⁵⁴⁾
 - 1310 New/worsening CNS Sx/findings ♦
 - 1320 Follow-up assessment⁽⁶⁷⁾

- 1400 Suspected CNS involvement with systemic disease **[One]**⁽⁵⁴⁾
 - 1410 Systemic lupus erythematosus (SLE)/vasculitis⁽⁵⁸⁾
 - 1420 HIV^(68, 69)
 - 1430 Neurofibromatosis (von Recklinghausen's disease)^(70, 71)
 - 1440 Tuberous sclerosis (Bourneville's disease)^(72, 73)
 - 1450 TB⁽⁷⁴⁾

- 1500 Multiple sclerosis (MS) **[One]**^(75, 76, 77, 78)
 - 1510 Suspected MS **[One]**^(79, 80, 81, 82, 83)
 - 1511 Clinically isolated syndrome (gadolinium contrast recommended) **[One]**⁽⁸⁴⁾
 - 1 Optic neuritis^(85, 86, 87)
 - 2 Ophthalmoplegia⁽⁸⁸⁾
 - 3 Transverse myelitis^(89, 90)
 - 1512 CNS deficit not in dermatomal/peripheral nerve distribution and other etiologies excluded **[One]**
 - 1 Sensory deficit
 - 2 Motor dysfunction
 - 1513 Loss of coordination and other etiologies excluded
 - 1520 Known MS with new/worsening symptoms (gadolinium contrast recommended)^(91*MDR)

- 1600 Suspected neurodegenerative disorder **[One]**
 - 1610 Adrenoleukodystrophy⁽⁹²⁾
 - 1620 Globoid (Krabbe's) leukodystrophy⁽⁹³⁾
 - 1630 Metachromatic leukodystrophy^(94, 95)

- 1640 Acute disseminated encephalomyelitis⁽⁹⁶⁾
- 1650 Alexander disease⁽⁹⁷⁾
- 1660 Canavan disease⁽⁹⁸⁾

- 1700 Suspected hydrocephalus **[One]**⁽⁹⁹⁾
 - 1710 Excessive rate of head growth
 - 1720 Bulging fontanel by PE
 - 1730 Papilledema by PE^(15, 16, 17)

- 1800 Nonacute onset mental status changes **[All]**⁽¹⁰⁰⁾
 - 1810 Sxs/findings **[One]**
 - 1811 Memory loss by Hx/PE⁽¹³⁾
 - 1812 Confusion/disorientation by Hx/PE⁽¹⁰¹⁾
 - 1813 Behavioral changes by Hx/PE
 - 1820 Lab results nondiagnostic for etiology of mental status change **[All]**
 - 1821 Na \geq 128 mEq/L(128 mmol/L)
 - 1822 Glucose \geq 60 and \leq 250 mg/dL(3.33 and $<$ 13.9mmol/L]
 - 1823 BUN \leq 80 mg/dL(28.6 mmol/L)
 - 1824 Ca \leq 11 mg/dL(2.75 mmol/L)
 - 1825 TSH normal
 - 1826 LFTs/ammonia normal
 - 1830 Urine drug/toxicology screen **[One]**
 - 1831 Negative
 - 1832 Not indicated

- 1900 Macrocephaly **[One]**^(102, 103)
 - 1910 Excessive rate of head growth
 - 1920 Focal neurologic finding by PE⁽¹⁹⁾

- 2000 Microcephaly^(104, 105)

Notes

(1)

CT and MRI each have relative advantages and disadvantages. CT, which tends to be better tolerated by patients, has the advantages of a shorter study time, better sensitivity for detecting acute hemorrhage, and excellent visualization of bony structures with less degradation of image quality by motion artifact. CT is often the preferred modality in rapidly evolving neurologic conditions (e.g., SAH, ICH) because it is widely available and may be performed easily in the setting of life support equipment. CT, however, poses the disadvantage of patient radiation exposure which carries an increased long-term risk of cancer. MRI tends to be more sensitive in detecting lesions in the brain, as well as assessment of cerebral ischemia. In acute stroke imaging, both CT and MRI are used to rapidly obtain the necessary anatomical, vascular, and functional information (Adam and Dixon, Grainger & Allison's Diagnostic Radiology: A Textbook of Medical Imaging 5th ed. 2008, 1936 p.).

(2)

Current magnetic resonance techniques lack ionizing radiation and provide images with high spatial resolution, excellent soft-tissue contrast, and multi-planar imaging capability (Widjaja and Raybaud, Neurosurg Focus 2008; 25(3): E3). Newer techniques include diffusion-weighted MRI and magnetic resonance spectroscopy. Diffusion-weighted imaging is being used for the evaluation of demyelinating disorders, presurgical evaluation and planning of brain tumors, and seizure disorders. Magnetic resonance spectroscopy is being used for evaluating many brain disorders, including brain tumors, leukodystrophies, and brain injuries (Abdelhalim and Alberico, Neurol Clin 2009; 27(1): 285-301, x; Provenzale, Emerg Radiol 2007; 14(1): 1-12).

(3)

The following are examples of relative and absolute contraindications to the use of magnetic resonance imaging:

- Implanted devices that are electrically or magnetically activated (e.g., cardiac pacemakers, automatic cardioverter defibrillators, drug infusion pumps, cochlear implants)
- Ferromagnetic metal objects (e.g., cerebral aneurysm clips, intraocular metallic foreign body, prostheses, screws)
- Pregnancy, first trimester
- Renal insufficiency in cases when magnetic resonance imaging is performed with gadolinium-based contrast

(4)

In children, the acute onset of persistent CNS findings may be due to lesions in the brain or spinal cord, inflammatory or vascular disorders, ischemic encephalopathy (e.g., near drowning), trauma, paroxysmal disease (e.g., seizures), or metabolic abnormalities. Ingestion of drugs and toxins can also cause acute neurologic symptoms (Lehman, Clinical Pediatric Emergency Medicine 2008; 9(2): 688-675; Eicher and Avery, Neurol Clin 2005; 23(2): 353-376). A recent study that looked at data sets for pediatric discharges with stroke, reported 3.70 per 100,000 hospitalizations for ischemic stroke and 2.37 per 100,000 for hemorrhagic stroke (Lo et al., J Child Neurol 2009; 24(2): 194-203). Stroke in children can be caused by AVM rupture, congenital or acquired heart disease, structural anomalies, infection, and hematologic abnormalities (Amlic-Lefond et al., Lancet Neurol 2008; 7(5): 425-435; Roach et al., Stroke 2008; 39(9): 2644-2691). Whether to perform CT or MRI is dependent on the child's clinical stability and is a matter of clinical judgement.

(5)

Sensory deficits commonly seen in stroke or TIA include hemianesthesia, single limb anesthesia, facial hemianesthesia or hypesthesia, and contralateral neglect (a neglect of the side opposite to the brain insult).

(6)

Motor deficits include motor weakness or paralysis (hemiparesis, quadriplegia, single limb involvement, or unilateral facial weakness), impaired coordination or ataxia, or dysphagia.

(7)

Language deficits most commonly present as aphasia or dysarthria. In the pediatric population, it may present as mutism.

(8)

Cognitive dysfunction may present as memory loss, confusion, disorientation, or behavioral changes.

(9)

Patients presenting with an acute onset of cognitive dysfunction should undergo imaging to search for potentially reversible structural causes of the mental status change, most notably subdural hematoma or acute hemorrhage (Christensen, Am J Emerg Med 2004; 22(3): 228-229). Acute cognitive changes are more likely the result of a toxic or metabolic disorder rather than structural pathology; however, these patients often cannot provide a clear history, making imaging, along with a metabolic evaluation, an important

element of the diagnostic algorithm.

(10)

Common visual symptoms include new vision loss, diplopia, visual field impairment and gaze impairment, and with TIA.

(11)

Altered levels of consciousness (e.g., coma, marked lethargy) may be due to a variety of mechanisms (e.g., increased ICP, infarct involving arousal systems).

(12)

Motor weakness occurring with a stroke or TIA tends to be focal (e.g., injury to a particular area of the brain results in a specific deficit).

(13)

In children, memory loss may present as declining school performance or regression in developmental milestones.

(14)

Imaging should be reserved for children who are at high risk of intracranial tumor or pathology (e.g., abnormal neurologic exam). Although traditionally CT has been the study of choice due to its availability and lower cost, MRI has recently emerged as the optimal initial study due to its capability to provide multiplanar views, its enhanced sensitivity in the detection of soft tissue changes, and its lack of ionizing radiation. However, since MRI requires more time and higher levels of sedation, CT is often obtained in rapidly evolving neurologic conditions in the pediatric population (Evans, *Neurol Clin* 2009; 27(2): 393-415). Whether to perform CT or MRI is a matter of clinical judgement.

(15)-DEF:

Papilledema is swelling of the optic disc, manifested by indistinct margins, hyperemia, venous engorgement, and lack of normal venous pulsations. Papilledema is a sign of increased ICP.

(16)

MRI is preferred for the evaluation of papilledema, but in cases where MRI is not available or the patient cannot tolerate the study, CT with contrast is a reasonable option.

(17)

Papilledema is not seen in neonates or infants with open fontanel; the increased ICP will cause bulging of the fontanel, sparing the optic disks. Older children with increased ICP and closed sutures may develop papilledema.

(18)

These criteria address a new or not as yet investigated headache. The symptom may actually have been present for some time before seeking medical attention. These criteria define risk factors which are worrisome for intracranial pathology (e.g., tumor).

(19)

Focal neurologic finding refers to a specific deficit that corresponds to a particular area of the brain (e.g., right arm weakness from a left motor cortex insult).

(20)-DEF:

Syncope is the transient loss of consciousness and postural tone caused by diminished cerebral blood flow characterized by rapid onset, short duration, and spontaneous complete recovery.

(21)

Mental status changes include confusion, lethargy, disorientation, somnolence, stupor, and coma.

(22)

Headaches causing patients to awaken from sleep may be caused by increased ICP.

(23)

Nocturnal vomiting may signify an intracranial tumor or may represent increased ICP.

(24)

Migraine is the most common type of chronic headache, triggered by factors such as stress, certain foods, hormonal changes, or lack of sleep. Treatment involves sleep, analgesics, and biobehavioral therapies (e.g., diet, relaxation techniques, cognitive therapies). Diagnosis of most patients with chronic headaches (e.g., migraine, tension headache) usually does not require imaging, particularly in the absence of neurologic signs and symptoms (Friedman and Grosberg, *Emerg Med Clin North Am* 2009; 27(1): 71-87; Lewis, *Neurol Clin* 2009; 27(2): 481-501; Pearlman, *Prim Care* 2004; 31(2): 407-415, viii).

(25)

Headache patterns of frequency and severity are often cyclical and related to environmental, hormonal, or social factors. Significant worsening of a previously stable headache can indicate new pathology and warrants imaging.

(26)

Seizures are categorized as either focal (partial) or generalized. Focal seizures can occur without impairment of consciousness (simple partial seizure) or with impairment of consciousness (complex partial seizure). Focal seizures can be associated with structural brain abnormalities; generalized seizures typically are not (Shneker and Fountain, *Dis Mon* 2003; 49(7): 426-478).

(27)

Seizure with accompanying neurologic findings may suggest an anatomic cause for the seizure (Adams and Knowles, *Am Fam Physician* 2007; 75(9): 1342-1347; Shneker and Fountain, *Dis Mon* 2003; 49(7): 426-478).

(28)

Neuroimaging is recommended in children with a postictal focal neurologic deficit that does not resolve, or when the child does not return to baseline neurologic function within several hours (Adams and Knowles, *Am Fam Physician* 2007; 75(9): 1342-1347).

(29)

Neuroimaging is recommended for the evaluation of patients presenting with a new onset seizure (Krumholz et al., *Neurology* 2007; 69(21): 1996-2007; ACEP Clinical Policies Committee, *Ann Emerg Med* 2004; 43(5): 605-625). MRI is the study of choice due to its enhanced sensitivity, although CT is a reasonable alternative when MRI is not feasible (Friedman and Sharieff, *Pediatr Clin North Am* 2006; 53(2): 257-277; Vattipally and Bronen, *Neuroimaging Clin N Am* 2004; 14(3): 349-372).

(30)

Seizures that occur in children with fever between the ages of 6 months and 5 years are called febrile seizures. Most are generalized and brief (lasting < 15 minutes) (American Academy of Pediatric (AAP), *Pediatrics* 2008; 121(6): 1281-1286). They may develop early in the course of an episode of otitis media or pharyngitis. Imaging is not necessary unless the child has accompanying focal neurologic findings.

(31)

Refractory seizures do not require imaging unless a new lesion is suspected (e.g., change in seizure pattern) or management will change based on the results of the scan.

(32)

Imaging is a crucial tool in the evaluation of children with refractory seizures. In some situations, advanced MR imaging techniques may be necessary to accurately localize the seizure foci. Newer imaging techniques include diffusion-weighted imaging, PET, and SPECT imaging. Further studies are needed however to evaluate their full efficacy (Widjaja and Raybaud, *Neurosurg Focus* 2008; 25(3): E3).

(33)

Refractory seizures in children may be treated surgically at a young age if a resectable seizure focus can be identified (Shneker and Fountain, *Dis Mon* 2003; 49(7): 426-478).

(34)

Time is required to adjust medications and assess their results before assuming that the seizures are refractory to treatment.

(35)

Prescription medications such as TCAs, antipsychotics, theophylline, and lidocaine can lower the seizure threshold. Attempts should be made to reduce or discontinue all such medications, but the risks and benefits of such an intervention need to be considered for each patient.

Recreational CNS stimulants (e.g., cocaine) can also cause seizures.

(36)

After head trauma, CT is the initial imaging modality of choice. It is widely available, fast, easy to obtain, and is capable of accommodating life-support and monitoring equipment (Slovis, *Caffey's pediatric diagnostic imaging* 11th ed. 2007, 3536). MRI may be more sensitive in assessing neural injury after the first 24 hours and for detecting small subdural hematomas. The utility of MRI for imaging head trauma may increase as technology such as magnetic resonance spectroscopy, diffusion-tensor imaging, and magnetization transfer MRI evolves (Jagoda et al., *Ann Emerg Med* 2008; 52(6): 714-748; Sigmund et al., *Pediatr Neurol* 2007; 36(4): 217-226; Smits et al., *Radiology* 2007; 245(3):831-838).

(37)

Clinical signs and symptoms of brain injury post trauma may be relatively insensitive indicators of intracranial injury in younger children, particularly those ≤ 6 months of age. A normal mental status or the absence of neurologic deficit does not exclude a serious brain injury (Thiessen and Woolridge, *Pediatr Clin North Am* 2006; 53(1):1-26, v). Infants < 3 months of age or who have a large scalp hematoma are especially at risk for skull fracture and intracranial injury. Imaging evaluation should, therefore, include children who are symptomatic after any head trauma but should also be directed to asymptomatic children who are 6 months of age or younger depending upon the child's clinical status.

(38)-DEF:

Retrograde amnesia is having no memory of the events that occurred before the trauma or condition.

(39)-DEF:

Anterograde amnesia is having no memory for events that occurred after the trauma or condition.

(40)

Vomiting after head trauma may be an early indication of increased ICP.

(41)

Clinical signs of skull fracture may include step-off or large hematoma, raccoon eyes, Battle's sign (post-auricular ecchymosis), hemotympanum, CSF otorrhea, rhinorrhea, or crepitance (Slovic, *Caffey's pediatric diagnostic imaging* 11th ed. 2007, 3536).

(42)

After head trauma, the imaging procedure of choice is a CT to assess for fracture, acute hydrocephalus, and to exclude intracranial hemorrhage. MRI may be more sensitive in assessing neural injury after the first 24 hours post trauma and for detecting small subdural hematomas. MRI technology is evolving with improvements in technology, such as magnetic resonance spectroscopy, diffusion-tensor imaging, and magnetization transfer MRI. This allows for decreased scan times and improvements in the sensitivity for detection of both structural and functional traumatic brain abnormalities. As MRI technology continues to evolve, there may be increased utility in head trauma imaging (Jagoda et al., *Ann Emerg Med* 2008; 52(6): 714-748; Sigmund et al., *Pediatr Neurol* 2007; 36(4): 217-226; Smits et al., *Radiology* 2007; 245(3):831-838; Metting et al., *Lancet Neurol* 2007; 6(8): 699-710).

(43)

Nonaccidental injuries, more commonly known as the "shaken baby syndrome" or the "shaking-impact syndrome" are largely restricted to children < 3 years of age. Risk factors include young parents, unstable family situations, low socioeconomic status, and disability or prematurity of the child (Christian, *Clin Fam Practice* 2003; 5(1): 21-46). Clinical features include retinal, subdural, and subarachnoid hemorrhage in an infant with little signs of external trauma (Gerber and Coffman, *Childs Nerv Syst* 2007; 23(5): 499-507).

(44)

Retinal hemorrhages are a cardinal manifestation of nonaccidental head trauma characterized by repetitive acceleration-deceleration forces with or without blunt head impact (shaken baby syndrome). Approximately 85% of affected children have retinal hemorrhage, with just under two thirds having extensive, "too numerous to count" multilayered hemorrhages extending out to the edges of the retina (Levin, *Pediatr Clin North Am* 2009; 56(2): 333-344). Although the presence of retinal hemorrhages does not confirm the diagnosis of shaken baby syndrome, studies report that retinal hemorrhages are common in abused children and rare in cases of accidental head injury in children under age 3 (Togioka et al., *Retinal hemorrhages and shaken baby syndrome: An evidence-based review*. 2008 [cited 2009 April 3]).

(45)

Fractures caused by abuse are more often multiple, bilateral, or associated with an intracranial subdural hematoma in comparison with those that result from accidental trauma (Kemp et al., *BMJ* 2008; 337: a1518).

(46)

Nonaccidental head injury is significantly more common when physical findings of traumatic head injury are present, and there is no history of trauma given. Imaging is essential to determine the extent of injury and to document the abuse (Schutzman and Greenes, *Ann Emerg Med* 2001; 37(1): 65-74).

(47)

CT is the preferred initial study for the child with suspected acute nonaccidental head trauma. Findings may include subdural hemorrhage, skeletal injuries and soft-tissue lesions. MRI may be used to further delineate the extent of injury, including subtle brain contusions, diffuse axonal injury, or loss of gray-white matter differentiation. MRI may also show involvement of the cervical spine or hemorrhage into the spinal canal as a result of the trauma (Reynolds, *Radiol Technol* 2008; 80(2): 151-170; Slovic, *Caffey's pediatric diagnostic imaging* 11th ed. 2007, 3536; Eltermann et al., *J Child Neurol* 2007; 22(2):170-175).

(48)

These criteria address CNS infection where the concern is generally for meningitis or encephalitis. Encephalitis generally presents with an altered sensorium in children. MRI is the preferred imaging modality as it is able to detect brain inflammation and edema in the cortex, the gray-white matter junction, the basal ganglia, or the cerebellum (Nichols and Rogers textbook of pediatric intensive care 4th edition ed. 2008, 1886 pages).

(49)

The superior contrast resolution of MRI makes it a more sensitive imaging tool for evaluating intracerebral abnormalities associated with a variety of complicated CNS infectious processes. Gadolinium contrast improves lesion delineation, localizes regions likely to provide positive biopsy, and identifies active disease. Additional information may be obtained from using diffusion-weighted imaging. In uncomplicated cases, CT may be obtained initially to identify patients at higher risk for herniation with intracranial abnormalities such as hydrocephalus, mass lesions, cerebral edema, and midline brain shift. LP often follows CT in the event the patient is considered low risk for herniation (Fitch et al., *Infect Dis Clin North Am* 2008; 22(1): 33-52, v-vi; Kastrup et al., *NeuroRx* 2005; 2(2): 324-332).

(50)

In the setting of meningitis or encephalitis, an immunocompetent host will usually demonstrate signs of infection such as fever, elevated WBC, neck stiffness, or neurologic signs. Imaging in this context is helpful to rule out other possible etiologies that might confound or complicate the diagnosis.

(51)-DEF:

Meningismus is a symptom complex associated with meningeal irritation, such as neck stiffness or a positive Kernig's or Brudzinski's sign (stretching of the nerve roots causes neck pain).

(52)

Meningismus may be seen with any meningeal irritant but should raise the suspicion of infectious meningitis.

(53)

Follow-up assessment is not necessary more frequently than every 7 to 10 days if the patient is stable or improving.

(54)

Whether to perform CT or MRI in this setting is a matter of clinical judgment.

(55)

Abscesses involving the CNS are uncommon. They sometimes result from direct trauma or neurosurgery, but may be caused by meningitis, rhinosinusitis, mastoiditis, and other extra-cranial sources (Ziai and Lewin, *Neurol Clin* 2008; 26(2): 427-468, viii).

(56)

Children with congenital heart disease (e.g., tetralogy of Fallot) are at risk of intracranial abscess from septic emboli (Ziai and Lewin, *Neurol Clin* 2008; 26(2): 427-468, viii; Overturf, *Pediatr Crit Care Med* 2005; 6(3 Suppl): S14-18).

(57)

A new or continued fever in a patient with a known abscess could indicate treatment failure or new primary infection.

(58)

The frequency of assessment is a matter of clinical judgment based upon the size, location, and number of intracranial abscesses. Follow-up is necessary to monitor resolution of the abscess in response to antibiotic therapy. If there is no progress, surgical drainage may be necessary.

(59)-RIN:

These criteria address a previously diagnosed brain tumor. For symptomatology which makes one suspect a new brain lesion, see the appropriate indication within this criteria subset.

(60)

Whether to perform a CT or MRI in this setting is a matter of clinical judgment. While MRI provides greater detail, CT is often sufficient for follow-up studies. It is generally best to perform the same study serially as this allows direct comparison of studies.

(61)

MRI (structural, diffusion weighted, or functional), PET scan, or magnetoencephalography are advanced imaging technologies that may be used to map pediatric brain tumors for interventional or operative planning (Nejat et al., *J Child Neurol* 2008; 23(10): 1136-1148).

(62)

If a single tumor has been identified by CT, MRI should be performed. MRI will better define the extent and characteristics of the isolated lesion and may show additional metastases. If the patient is not a surgical candidate because of other widespread metastases, MRI may not be necessary.

(63)

The interval for periodic assessment in stable patients is a matter of clinical judgment. Studies are generally performed no more frequently than every two cycles of chemotherapy.

(64)

The post treatment assessment is generally performed 4 to 6 weeks after therapy is completed.

(65)-DEF:

An arteriovenous malformation (AVM) is a vascular lesion consisting of dilated feeding arteries and a core of tangled vascular loops that terminate in draining veins.

(66)

Treatment of brain AVM's in children depends on the size, venous drainage pattern (superficial or deep), and the location of the AVM (Jordan and Hillis, *Pediatr Neurol* 2007; 36(2):73-80).

(67)

It may be reasonable to obtain imaging studies in the immediate postoperative period following an intracranial procedure. These patients are often acutely ill and the determination of neurologic status can be difficult, as it is often complicated by sedating medication or cerebral edema. Clinical judgment may dictate early scanning if the patient fails to progress as expected, even if worrisome focal neurologic signs are absent.

(68)

Symptoms and findings of CNS involvement by SLE or vasculitis are quite varied but typically include headache, mental status changes, seizure, or stroke. Focal or nonspecific neurologic findings may also accompany HIV, without concomitant opportunistic CNS infection. These diseases have characteristic imaging findings that aid in diagnosis and thereby guide therapy.

(69)

HIV in children is most often acquired by vertical transmission in utero, often with direct involvement of the brain by HIV. Characteristic MRI findings include cerebral atrophy, white matter abnormalities, and basal ganglia calcification (Hunter and Morriss, *Semin Pediatr Infect Dis* 2003; 14(2): 140-164).

(70)-DEF:

Type I neurofibromatosis (von Recklinghausen's disease) is a disorder characterized by cafe au lait spots, neurofibromas, freckling in the axillary or inguinal areas, optic gliomas, and osseous lesions. It most commonly presents with cognitive or psychomotor problems, learning disabilities, or mental retardation.

(71)

Brain imaging in neurofibromatosis is performed with special cuts through the optic nerves to exclude optic gliomas which can occur in up to 15% of patients. Baseline MRI or CT provides information on growth characterization of the tumor and assists in plans for treatment (e.g. chemotherapy) (Williams et al., *Pediatrics* 2009; 123(1): 124-133).

(72)-DEF:

Tuberous sclerosis (Bourneville's disease) is an inherited disease of unknown etiology. The classic triad of seizures, mental retardation, and sebaceous adenomas is seen in only 1/3 of patients. Tuberous sclerosis typically presents with a wide variety of symptoms, from simple skin findings (e.g., facial angiofibromas) to seizure disorders with mental retardation later in life.

(73)

CT findings in tuberous sclerosis include pathognomonic subependymal calcifications, widened gyri, and brain tumors. MRI often detects hypomyelinated lesions that are associated with tubers or developmental abnormalities of the cerebral cortex, which are present in over 80% of patients. Tubers can calcify or undergo cystic degeneration. Molecular genetic testing of the TSC1 or TSC2 loci is currently viewed as corroborative (Crino et al., *N Engl J Med* 2006; 355(13): 1345-1356; Kandt, *Neurol Clin* 2003; 21(4): 983-1004).

(74)

Although the lungs are the primary site of infection in TB, approximately 10% of immunocompromised patients will develop CNS involvement. The most severe CNS complication is the development of tuberculous meningitis (Thwaites and Tran, *Lancet Neurol* 2005; 4(3): 160-170).

(75)

Multiple sclerosis (MS) is a chronic inflammatory disease of the CNS. The natural history of MS is characterized by the relapse and remission of various focal symptoms; some patients experience a chronic progressive pattern of disability (Courtney et al., *Med Clin North Am* 2009; 93(2): 451-476; Birnbaum, *Adv Neurol* 2006; 98: 111-124). Sites of autoimmune mediated demyelination cause focal neurologic impairment, which may correlate with MRI signal intensity changes within the white matter. The hallmark of MS lesions is their bright appearance on T2-weighted images in the brain; lesions are also commonly seen in the spinal cord (Simon, *Radiol Clin North Am* 2006; 44(1): 79-100; Bakshi et al., *Neurology* 2004; 63(11 Suppl 5): S3-11). The use of MRI has allowed earlier confirmation of the diagnosis, resulting in earlier medical intervention and improved management of the disease. LP results and visual evoked potentials can suggest the diagnosis.

(76)

Less than 10% of MS patients are under the age of 18. The average age of onset of early onset, or juvenile, MS is between 8 and 14 years of age. Children typically present with a higher incidence of cerebellar or brainstem findings and a lower incidence of spinal cord symptoms than adults. MS in children appears to follow a less aggressive course than in adult patients, although nearly 15% of MS cases in children are progressive from the onset. Children and adolescents take longer to convert to secondary progressive disease but do so at an age 10 years younger than adult patients (Banwell et al., *Lancet Neurol* 2007; 6(10): 887-902; Renoux et al., *N Engl J Med* 2007; 356(25): 2603-2613; Ness et al., *Neurology* 2007; 68(16 Suppl 2): S37-45).

(77)

MRI is primarily used for the evaluation of suspected MS, as well as for following new or worsening symptoms. MRI can exclude other conditions that would account for the patient's symptoms and exam findings, can establish the presence of clinically silent lesions, and can demonstrate new lesions (Royal College of Physicians, *Multiple Sclerosis. National clinical guideline for diagnosis and management in primary and secondary care. 2004, 197*). CT is not indicated as a diagnostic test for suspected MS.

(78)

Functional, magnetization transfer, diffusion tensor, and spectroscopy MRI are now being used outside clinical trials as adjunctive measures for diagnosing and monitoring disease progression and treatment response (Ali and Buckle, *Neurol Clin* 2009; 27(1): 203-219, ix; Bakshi et al., *Lancet Neurol* 2008; 7(7): 615-625; Rovira and Leon, *Eur J Radiol* 2008; 67(3): 409-414; Fazekas et al., *J Neuroimaging* 2007; 17 Suppl 1: 50S-55S).

(79)

MS can present with sensory deficits, motor dysfunction, or cerebellar or brainstem dysfunction. There is usually no set pattern to the symptoms (Lublin, *Neurol Clin* 2005; 23(1): 1-15).

(80)

There are a number of diseases that may present in a similar manner to MS. These include acute disseminated encephalomyelitis, CNS vasculitis, migraine, tumor, sarcoidosis, Lyme disease, Sjogren's syndrome, SLE, and vitamin B₁₂ deficiency (Courtney et al., *Med Clin North Am* 2009; 93(2): 451-476; Miller et al., *Mult Scler* 2008; 14(9): 1157-1174; Krupp et al., *Neurology* 2007; 68(16 Suppl 2): S7-12; Birnbaum, *Adv Neurol* 2006; 98: 111-124). Many of these diagnoses can be ruled out with laboratory testing (e.g., CBC, ANA, ESR, vitamin B₁₂, TSH).

(81)

Gadolinium contrast is used to identify any disruption of the blood-brain barrier secondary to active inflammation. The number of enhancing lesions is the most clinically relevant measure of ongoing disease activity (Simon, *Radiol Clin North Am* 2006; 44(1): 79-100, viii; Bakshi et al., *Neurology* 2004; 63(11 Suppl 5): S3-11).

(82)

MRI is considered positive for MS if the following criteria are met (Frohman et al., *Neurology* 2003; 61(5): 602-611; McDonald et al., *Ann Neurol* 2001; 50(1): 121-127; Tintore et al., *AJNR Am J Neuroradiol* 2000; 21(4): 702-706; Barkhof et al., *Brain* 1997; 120 (Pt 11): 2059-2069):

For dissemination in space, 3 out of 4 of the following are found:

- 1 gadolinium-enhancing brain or spinal cord lesion *or* 9 T2 brain or cord lesions if there is no gadolinium-enhancing lesion
- ≥ 1 infratentorial brain or cord lesion
- ≥ 1 juxtacortical lesion
- ≥ 3 periventricular lesions

For dissemination in time:

- The appearance on a subsequent MRI (≥ 3 months from the previous MRI) of a new T2 or gadolinium-enhancing lesion at a site different from the initial event

Newer criteria have been proposed that include dissemination in space of at least one T2 lesion in each of at least 2 locations: juxtacortical, periventricular, infratentorial, or spinal cord. The dissemination in time requires a new T2 lesion on a follow-up scan (Swanton et al., *Lancet Neurol* 2007; 6(8): 677-686). The modified criteria are highly specific (93%) and more accurate for the clinical development of MS than the McDonald criteria (Swanton et al., *J Neurol Neurosurg Psychiatry* 2006; 77(7): 830-833).

(83)

Children with MS may not meet the McDonald criteria since they often have less dissemination in space (smaller number of lesions); MRI features do not appear the same in children as they do in adults with MS (Banwell et al., *Lancet Neurol* 2007; 6(10): 887-902; Krupp et al., *Neurology* 2007; 68(16 Suppl 2): S7-12).

(84)

Patients may present with clinically isolated syndrome (CIS) or a monosymptomatic attack. These attacks last at least 24 hours and consist of symptoms of optic neuritis, brain stem syndrome (e.g., internuclear ophthalmoplegia), or spinal cord syndrome (e.g., partial transverse myelitis, hyperreflexia, decreased motor, bowel, or bladder control) (Miller et al., *Mult Scler* 2008; 14(9): 1157-1174; Simon et al., *AJNR Am J Neuroradiol* 2006; 27(2): 455-461).

(85)-DEF:

Optic neuritis is an inflammation of the optic nerve. Symptoms include pain in and around the eye, altered visual acuity (e.g., blurred vision), and altered color perception.

(86)

A higher risk for developing future demyelination is seen in young females presenting with unilateral or painful optic neuritis, along with the finding of MRI abnormalities at the time of the attack (The Optic Neuritis Study Group, *Arch Neurol* 2008; 65(6): 727-732; Miller et al., *Mult Scler* 2008; 14(9): 1157-1174; Thrower, *Neurology* 2007; 68(24 Suppl 4): S12-15).

(87)

Several small studies suggested increased risk for the subsequent development of MS in children with bilateral optic neuritis, lesions of the optic pathways on MRI, or a family history of optic neuritis (Banwell et al., *Lancet Neurol* 2007; 6(10): 887-902; Ness et al., *Neurology* 2007; 68(16 Suppl 2): S37-45).

(88)-DEF:

Ophthalmoplegia is paralysis of the eye muscles.

(89)-DEF:

Transverse myelitis is inflammation (leading to demyelination) involving the full diameter of the spinal cord but limited in longitudinal extent.

(90)

The risk of developing subsequent MS is highest when the patient presents with asymmetric, incomplete transverse myelitis (Miller et al., *Mult Scler* 2008; 14(9): 1157-1174; Thrower, *Neurology* 2007; 68(24 Suppl 4): S12-15).

(91)-MDR:

Because conventional MRI does not show remyelination or the pathophysiology of lesions well, there is a mismatch between symptomatology and MRI findings (Zivadinov et al., *J Neurol* 2008; 255 Suppl 1: 61-74). Currently, conventional MRI is not indicated for routine follow-up of patients with known MS unless the patient exhibits a clinical change. A new lesion by imaging may not reflect treatment failure but may be a manifestation of the natural history of the disease (Simon, *Radiol Clin North Am* 2006; 44(1): 79-100, viii; Filippi et al., *Eur J Neurol* 2006; 13(4): 313-325). Studies are ongoing regarding the correlation of MRI activity with relapse rate. The evidence varies as to whether MRI should be used to monitor treatment, rather than waiting for relapses and changes in clinical symptomatology (Sormani et al., *Ann Neurol* 2009; 65(3): 268-275). Until solid results point to the use of MRI for routine follow-up, requests for MRI without a clinical change require secondary medical review.

(92)-DEF:

Adrenoleukodystrophy is inherited as an X-linked recessive disorder, presenting in boys between the ages of 5 and 10 years. This disorder is characterized by adrenocortical insufficiency, skin hyperpigmentation, progressive dementia, and spasticity. MRI shows periventricular demyelination.

(93)-DEF:

Globoid (Krabbe's) leukodystrophy is a metabolic encephalopathy of infancy with rapidly progressive cerebral degeneration, myelin loss, and infiltration of the white matter with globoid cells. Children present early with seizures, spasticity, and difficulty feeding.

(94)-DEF:

Metachromatic leukodystrophy is a metabolic disorder characterized by myelin loss and accumulation of lipids in the white matter of the central and peripheral nervous system. Patients develop mental retardation and progressive paralysis. The diagnosis is confirmed by enzymatic studies on leukocytes.

(95)

The onset of the infantile form of metachromatic leukodystrophy is usually by age 2, with juvenile onset occurring between 5 to 10 years of age. Initial findings include ataxia, neuropathy, and decreased or absent muscle stretch reflexes. The clinical course rapidly progresses over a 1 to 2 year time period, with the development of spasticity, loss of intellectual abilities, optic atrophy, and seizures. Life expectancy varies with the age of disease onset. Death before age 10 is typical for those with infantile-onset disease; those with juvenile onset may live until their late teens or early twenties (Kliegman and Nelson, Nelson textbook of pediatrics, 18th ed. 2007, lli, 3147 p.; Crumrine, *Pediatr Rev* 2001; 22(11): 370-379).

(96)

Acute disseminated encephalomyelitis is an inflammatory demyelinating disease of presumed autoimmune origin that develops 2 to 3 weeks following a viral infection or vaccination. The median age of onset is from 4 to 8 years. The patient presents most commonly with seizures, fever, acute hemiparesis, ataxia, and an altered level of consciousness. MRI shows multiple ill-defined lesions with basal gray matter involvement, consistent with asymmetric demyelination of the brain stem, cerebellum, and cerebrum (Noorbakhsh et al., *Neurol Clin* 2008; 26(3): 759-780, ix).

(97)

Alexander disease is a rare disorder which causes progressive macrocephaly during the first year of life. Children develop progressive loss of intellect, spasticity, and unresponsive seizures with death occurring by age 5. Imaging shows white matter degeneration that prominently affects the frontal lobes of the brain (van der Knaap et al., *Neurology* 2006; 66(4): 494-498).

(98)

Canavan disease, an autosomal recessive disorder, is caused by the deficiency of the enzyme aspartoacylase, which, in turn, leads to N-acetylaspartic acid buildup in the white matter of the brain. An infant may appear normal at birth, with symptoms of progressive macrocephaly, severe hypotonia, and persistent head lag developing by 3 to 6 months of age. The clinical course rapidly develops over the next 2 to 3 years with findings of hyperreflexia, seizures, optic atrophy, and swallowing difficulties. Imaging reveals white matter degeneration of the brain primarily in the cerebellum and brainstem (Kliegman and Nelson, Nelson textbook of pediatrics, 18th ed. 2007, lli, 3147 p.).

(99)-DEF:

Hydrocephalus is an excessive accumulation of cerebrospinal fluid intracranially often resulting in ventricular dilation and increased ICP.

(100)

Diagnostic evaluation of the child with mental status changes typically involves identification and rapid elimination of hypoglycemia, abnormalities of electrolytes or renal function, and intoxicants.

(101)

Confusion is characterized by fluctuating levels of consciousness and attention. Most commonly, confusion has a toxic or metabolic etiology, but imaging may be required to exclude a structural cause.

(102)-DEF:

Macrocephaly is defined as a head circumference >2 SD above the mean for age and sex.

(103)

Imaging for patients with macrocephaly is indicated when there is focal neurologic findings or excessive head growth which could suggest increased ICP most likely caused by hydrocephalus, fluid collections, or neoplasm. Imaging is not needed if the head circumference is large but growing according to the growth curves and not at an accelerated rate. Macrocephaly with normal head growth implies familial macrocephaly. Imaging permits accurate diagnosis and prognosis, serves as a basis for comparison as the child grows, and is performed to define any structural cause of the macrocephaly that may be amenable to operative repair (e.g., brain tumor, arachnoid cyst) (Adam and Dixon, *Allison's diagnostic radiology : a textbook of medical imaging*, 5th ed. 2008, 950p, 174p; Medina et al., *AJNR Am J Neuroradiol* 2001; 22(3): 564-570).

(104)-DEF:

Microcephaly is defined as a head circumference > 3 SD below the mean for age and sex.

(105)

Microcephaly should be suspected in full-term newborns and infants up to 6 months of age whose chest circumference exceeds head circumference (unless the child is very obese). Imaging permits accurate diagnosis and may demonstrate calcifications, malformations, or atrophic patterns that suggest congenital infections (e.g., TORCH, Coxsackie B virus) or genetic syndromes (e.g., trisomy 13, 18, 21) that can cause microcephaly (Adam and Dixon, *Allison's diagnostic radiology : a textbook of medical imaging*, 5th ed. 2008, 950p, 174p). MRI can also help to determine a specific diagnosis in patients with microcephaly and global developmental delay (Shevell et al., *Neurology* 2003; 60(3): 367-380).