NEUROIMAGING FOR THE GENERAL NEUROLOGIST II: BRAIN AND SPINE

Emergency and Critical Care Neuroimaging

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The primary goals of neuroimaging in the emergency or critical care setting is to assess the state and viability of cerebral or spinal tissue after the onset of neurological symptoms, and to establish a clinico-radiographic correlation for diagnosing and monitoring the course of a disease process and response to therapeutic interventions. In practice, neuroimaging is used to determine whether emergent medical therapy (e.g., thrombolysis in acute stroke) or surgical therapy (e.g., hemicraniectomy for malignant edema) is required, and to assess whether the damage to the brain or spinal cord is ongoing or complete (e.g., is there additional tissue "at risk"). One should always try to determine the primary injury, the presence of secondary problems (e.g., obstructive hydrocephalus resulting from cerebral edema), and the likelihood of secondary problems to arise in the future. With all of this information, imaging can be used in conjunction with clinical data to help direct care and prognosticate outcome.

CT is widely available and the most useful technique for rapidly assessing the patient with neurologic symptoms that have begun abruptly. The principal concerns are hemorrhage, infarction, trauma, and mass (including tumor, abscess, edema, etc.). One can assess whether an abnormality is focal or diffuse, and whether it involves the epidural, subdural, subarachnoid, intraparenchymal, or intraventricular compartments. Next, a determination of whether the involved tissue volume is increased or decreased as well as whether the involved tissue is abnormally bright (hyperdense) or dark (hypodense), can help refine a differential diagnosis.

MRI is considerably more sensitive for detecting subtle injury to the brain or spinal cord such as diffuse axonal injury, small infarctions, and encephalitis, but in the emergency setting assessment of tissue volume and signal as well as the effect of a lesion on adjacent structures (e.g. compression, herniation, hydrocephalus) are of greatest significance.

Beyond hemorrhage and ischemia, many other processes can cause acute alteration in arousal and require critical care management. The presence of other clinical features such as fever, seizures, myoclonus, preceding illness, a history of immunosuppression, certain medication exposures, and certain metabolic disturbances, can suggest particular dramatic and severe neurological syndromes including limbic encephalitis (viral or paraneoplastic), Wernicke encephalopathy, acute disseminated encephalomyelitis (ADEM), prion disease, progressive multifocal leukoencephalopathy (PML), posterior reversible encephalopathy syndrome (PRES), reversible cerebral vasoconstriction syndrome (RCVS), convulsive or non-convulsive status epilepticus, serotonin syndrome, neuroleptic malignant syndrome, acute hepatic encephalopathy, and pontine (and extra-pontine) myelinolysis. Many of these syndromes have fairly characteristic imaging features.

Herniation refers to the abnormal movement or position of brain or spinal cord contents. Intracranially, there are several types of herniation including uncal (transtentorial descending, usually caused by temporal lobe masses), central (descending, usually caused by midline supratentorial masses), subfalcine (usually caused by hemispheric masses), transcalvarial (usually caused by traumatic or surgical craniotomy or craniectomy), transtentorial (ascending, usually caused by a superior cerebellar mass), and tonsillar (descending, usually caused by an inferior cerebellar mass). Herniation can also rarely occur in the spinal canal, where a segment of cord protrudes through a defect in the dura, with variable clinical symptomatology.

Acute compression of the spinal cord or cauda equina is a neurological emergency. X-ray of the spine can easily demonstrate vertebral misalignments and fractures, whereas CT provides better visualization of intervertebral discs and neural elements. MRI provides the highest resolution images of

the spine and is particularly helpful when the mechanism of cord compression is obscure, for example, when it occurs in the absence of trauma, or when other pathophysiologies such as infectious or non-infectious inflammation or neoplasia are suspected. Though no evidence-based guidelines or standards exist regarding timing of spinal cord or cauda equina decompression surgery or optimal surgical strategy, generally, incomplete cord injuries are decompressed early (within 24h).

References

Fink KR, Benjert JL. Imaging of nontraumatic neuroradiology emergencies. Radiol Clin North Am. 2015;53(4):871-90.

Ropper AH, Gorson KC. Concussion. N Engl J Med. 2007;356(2):166-72.

Rincon S, Gupta R, Ptak T. Imaging of head trauma. Handb Clin Neurol. 2016;135:447-77.

Edlow BL, Wu O. Advanced neuroimaging in traumatic brain injury. Semin Neurol. 2012;32(4):374-400.

Edlow BL, Haynes RL, Takahashi E, et al. Disconnection of the ascending arousal system in traumatic coma. J Neuropathol Exp Neurol. 2013;72(6):505-23.

Lin MP, Liebeskind DS. Imaging of Ischemic Stroke. Continuum. 2016;22(5):1399-423.

Hakimi R, Garg A. Imaging of Hemorrhagic Stroke. Continuum. 2016;22(5):1424-50.

Greer DM, Wu O. Neuroimaging in Cardiac Arrest Prognostication. Semin Neurol. 2017;37(1):66-74.

Klein JP. Neuroimaging for the clinician. In "ACP Medicine, a publication of the American College of Physicians", Ed. EG Nabel. Decker Publishers, 2012.

Klein JP. Imaging of progressive weakness or numbness of central or peripheral origin. Handb Clin Neurol. 2016;136:923-37.

Klein JP. Imaging of noninfectious inflammatory disorders of the spinal cord. Handb Clin Neurol. 2016;136:733-46.

Klein JP. A practical approach to spine imaging. Continuum. 2015;21(1):36-51.