Supplementary Table 3. Neuroimaging findings in specific clinical encephalopathy syndromes

Imaging techniques / References	Total no. of patients	Neuroimaging findings	Predictive value for poor outcome
Hepatic encephalopathy			
CT 1 case series [1]	17 patients	Normal	No clear evidence
MRI 1 prospective study [2] 4 retrospective studies [3-6] 5 case series [1,7-10] 1 case report [11]	290 patients	- T1 hyperintensities in the globus pallidus and, less frequent, in the substantia nigra and the midbrain tegmentum - FLAIR and DWI hyperintense thalami, posterior limbs of the internal capsule, periventricular region, dorsal brain stem, and diffuse cortical involvement in 1 study of 20 patients - Connectivity: decreased in the caudate of the anterior/middle cingulate gyrus; increased in the caudate of the left motor cortex; reduced between the putamen and the anterior cingulate gyrus, right insular lobe, inferior frontal gyrus, left parahippocampal gyrus, and the anterior lobe of the right cerebellum; increased between the putamen and right middle temporal gyrus	Correlations between the cortico-striatal connectivity and neuropsychological performances, but not between the striatal connectivity and globus pallidus signal intensity
MRS 2 case reports [10,12]	2 patients	- Increased glutamate/glutamine ratio and low myoinositol and choline - Diminished choline and elevated glutamate/glutamine ratio in the parietooccipital cortex	No clear evidence
SPECT 2 case reports [13,14]	2 patients	- High blood flow in the cerebellum, basal ganglia and cerebral cortex - Alteration of striatal D2-receptor binding and dopamine re-uptake	No clear evidence
PET 1 prospective study [15] 1 case report [16]	41 patients	- Hypoperfusion of the superior and middle frontal gyri, and inferior parietal lobules - Increased expression of peripheral benzodiazepine binding sites prefrontal and striatal in cirrhotic patients	No clear evidence
Wernicke's encephalopathy			
CT 1 retrospective study [17] 1 case series [18] 4 case reports [19-22]	22 patients	Hypodense paraventricular thalamic regions with or without contrast enhancement and, less frequent, hypodense periaqueductal regions, tectum of the midbrain, and tegmentum of the pons	No clear evidence
MRI 1 prospective study [23] 4 retrospective studies [17,24-26] 2 case series [18,27] 5 case reports [21,22,28-30]	202 patients	- T2 and FLAIR hyperintense periaqueduct and medial thalamic regions. Less frequent hyperintense mamillary bodies, periaqueductal region, hypothalamus, tectum, and cerebellum - In 1 study 78% of patients with Wernicke's encephalopathy had smaller mamillary bodies than controls - Contrast-enhanced mamillary bodies were related to alcohol abuse - Atrophic mammillary bodies and cerebellar vermis (chronic phase)	No clear evidence
MRS 2 case reports [30,31]	2 patients	Thalamic lactate increase and low N-acetyl-aspartate/creatine	No clear evidence
SPECT 1 case report [32]	1 patient	Hypoperfusion fronto-parietal and in the right basal ganglia	No clear evidence
Posterior reversible encephalopathy			
CT 1 retrospective study [33] 2 case reports [34,35]	69 patients	- Hypodensities in the parietooccipital subcortical white matter and cerebellum with increased cerebral blood volume, blood flow, and reduced time to peak mainly in the posterior vascular distribution (Features of PRES in 45%, unspecific in 33% and normal in 22%) - In patients with PRES on MRI, CT was negative/unspecific in 66%	No clear evidence
MRI 4 retrospective studies [33,36-38] 2 case series [39,40]	355 patients	- T2, FLAIR, and DWI hyperintensities in the posterior circulation areas and, less frequent, in the anterior circulation structures. ADC values in areas of abnormal T2 signal were high	More extensive T2 signal abnormalities were seen with poor

7 case reports [11,34,41-45]		- Contrast-enhancement, restrictions on DWI and ADC	outcome
MRS 2 case series [39,46] 1 case report [41]	13 patients	Decrease in N-acetyl-aspartate in patients with normal MRI or reversible MRI changes and only minimal elevation of choline	No clear evidence
Acute disseminated encephalomyelitis			
CT 11 retrospective study [47]	12 patients	Normal	No clear evidence
MRI 2 retrospective studies [47,48] 1 prospective study [49]	50 patients	- T2 and FLAIR with multiple brain lesions in the deep and subcortical white matter and in 1/3 in the brainstem and spinal cord with contrast enhancement - In the first week (acute phase) DWI with restricted diffusion, and later (subacute phase) with increased diffusion	No clear evidence
MRS 1 retrospective study [48]	8 patients	Decreased N-acetylaspartate in regions corresponding to the T2 signal intensity in the subacute phase	No clear evidence
Paraneoplastic limbic encephalitis			
CT 5 case reports [50-54]	5 patients	Normal	No clear evidence
MRI 2 retrospective studies [55,56] 10 case reports [50-54,57-61]	83 patients	- T2 and FLAIR hyperintensities with mesial temporal contrast enhancement in >50% and/or atrophy - Subcortical regions, the cerebellum or brainstem may be involved	No clear evidence
PET 4 case reports [50,58,62,63]	4 patients	PET may reveal increased metabolism mesial temporal	No clear evidence
Autoimmune limbic encephalitis			
CT 4 case reports [64-67]	4 patients	Normal	No clear evidence
MRI NMDA 1 retrospective study [68] 1 case series [69] 9 case reports [65,66,70-76] SREAT 4 retrospective studies [77-80] 10 case reports [67,81-87]	158 patients	- In NMDAR-antibody mediated limbic encephalitis, MRI is mostly (50%) normal but can show T2 and FLAIR hyperintensities temporal and rarely extratemporal. MRI is used to exclude other causes of encephalopathy - In SREAT, T2 and FLAIR can rarely resemble acute demyelinating encephalomyelitis or show hippocampal or multifocal hyperintensities	No clear evidence
MRS 2 case reports [82,88]	2 patients	In SREAT, decreased N-acetyl-aspartate, myo-inositol peaks, elevations in lipid, lactate, glutamate/glutamine and choline peaks support inflammation	No clear evidence
SPECT 1 retrospective study [79] 4 case reports [89-92]	16 patients	- In NMDAR-antibody mediated limbic encephalitis, abnormal multifocal cerebral blood flow - In SREAT, decreased tracer uptake in the striatum and global cortical hypoperfusion	No clear evidence
Herpes simplex encephalitis			
CT 3 retrospective studies [93-95] 1 case series [96] 2 case reports [97,98]	121 patients	Usually normal, but can characteristically show reduced attenuation in the temporal lobes after the first week of the disease	Lesions on CT are predictive of prolonged course of disease
MRI 5 retrospective studies [95,99-102] 4 case series [96,103-105] 22 case reports [97,98,106-125]	298 patients	- T2, FLAIR, and DWI hyperintensities in the medial temporal lobes, the orbital surface of the frontal lobes, the insular cortex, the angular gyrus, and in the insulas early in the course. Rarely the thalami and insulas may be involved. - Abnormal areas may show enhancement with gadolinium - Midline shift may be present with large cerebral edema	The extent of brain involvement is an independent risk factor for poor prognosis
SPECT 1 case series [96] 1 case report [97]	3 patients	Increased tracer accumulation which reflects hyperperfusion possibly earlier than pathologic signals appear on MRI	No clear evidence
Susac's syndrome			

CT 1 case report [126]	1 patient	Does not reveal any oft he specific structural abnormalities, but can demonstrated foci of subtle low attenuation in the corpus callosum	No clear evidence
MRI 4 case series [127-130] 12 case reports [126,131-141]	53 patients	- T2 and FLAIR hyperintensities in the corpus callosum, which is always involved - Any part of the corpus callosum can be involved, but predominately the central fibers are showing microinfarcts that are typically small but may sometimes be large - Foci in the corpus callosum may enhance following gadolinium administration and there can be restricted diffusion with corresponding low signal intensity on the ADC map - Spinal cord involvement is rare but exists - Subsequently, central callosal holes arise	No clear evidence
PET 1 case report [142]	1 patient	Marked hypometabolism in the frontal, parietal and temporal lobes – an unspecific pattern that can be mistaken as ADEM	No clear evidence
Creutzfeldt-Jakob disease			
CT 3 case series [143-145]	11 patients	Non-specific generalized cortical and subcortical atrophy in the later phases of disease	No clear evidence
MRI 13 retrospective studies [146-158] 12 case series [144,159-169] 26 case reports [170-197]	3393 patients	- T2 and FLAIR hyperintensities in the cerebral cortex and lesions in the putamen and caudate head isointense to cortex - Less frequently, hyperintensity can be detected in the globus pallidus, thalamus, the deep white matter, and the cerebral and cerebellar cortex. Laminar lesions may be observed in the cerebral cortex and cerebellum - DWI is most sensitive in early stages uncovering the altered diffusion in the regions mentioned above - In vCJD symmetrical hyperintensities of the pulvinar thalami (relative to the cortex and especially the anterior part of the putamen) are characteristic and known as the “pulvinar sign”	Patients with cortical plus basal ganglia hyperintensity have shorter interval from symptom onset to akinetic mutism than those with isolated cortical ribbon hyperintensity
MRS 1 case report [198]	1 patients	Decreased N-acetyl-aspartate and slightly increased levels of myoinositol in the striatum and the insular cortex.	No clear evidence
SPECT 1 case report [172]	1 patient	Hypoperfusion in the cerebral cortex	No clear evidence
PET 1 case series [151] 1 case report [176]	15 patients	Hypometabolism in the cerebral cortex and the basal ganglia	No clear evidence

CT = computed tomography; MRI = magnetic resonance imaging; DWI = diffusion-weighted imaging; FLAIR = fluid attenuated inversion recovery; MRS = magnetic resonance spectroscopy; SPECT = single photon emission computed tomography; PET = positron emission tomography; ADEM = acute disseminated encephalomyelitis; NMDAR = N-methyl-D-aspartate receptor; SREAT = steroid responsive encephalitis with autoimmune thyroiditis

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