Supplementary Table 2. Neuroimaging findings in encephalopathies with specific biochemical derangements

Imaging techniques / References	Total no. of patients	Neuroimaging findings	Predictive value for poor outcome
Uremic encephalopathy			
CT 2 case series [1,2] 2 case reports [3,4]	7 patients	Hypodense basal ganglia and capsules	No clear evidence
MRI 3 case series [1,2,5] 6 case reports [3,4,6-9]	15 patients	- T2, FLAIR and DWI hyperintense basal ganglia, capsules and inconsistently in cortical areas - Additional involvement of white matter and cerebral peduncles, occipital lobes and thalami	No clear evidence
MRS 1 case report [10]	1 patient	Decrease of N-acetyl-aspartate and the presence of lactate	No clear evidence
PET 1 case series [11]	2 patients	Decreased glucose metabolism in basal ganglia	No clear evidence
Hyperammonemic encephalopathy			
MRI 2 case series [12,13]	7 patients	T2, FLAIR and DWI hyperintense cortical areas of the insula and the cingulum	No clear evidence
MRS 2 case reports [14,15]	2 patients	Increased glutamine and glutamate; low myoinositol and choline	No clear evidence
Hypo-/hypernatremic encephalopathies (pontine or extrapontine myelinolysis)			
CT 2 retrospective studies [16,17] 1 Case series [18]	48 patients	Normal in a few patients and hypodense pontine lesions in the others	No clear evidence
MRI 2 retrospective studies [16,17] 6 case series [18-23] 9 case reports [4,15,24-30]	136 patients	- T2, FLAIR and DWI hyperintense lesions of the pons. Less frequent lesions of the thalamus, midbrain, cortical gray matter, hippocampus, caudate, putamen, and middle cerebral peduncle - In 1 patient T2 hyperintensities characteristically were distributed along the crowns and sides of the cerebral gyri	No correlation between the lesion size and outcome
SPECT 2 case reports [31,32]	2 patients	Decreased striatal dopamine transporter binding and pontine hyperperfusion during recovery from pontine myelinolysis	No clear evidence
Hypoglycemic encephalopathy			
CT 1 case series [33] 1 case report [4]	5 patients	Enhancing, hypodense basal ganglia, cerebral cortex, hippocampus, and substantia nigra	No clear evidence
MRI 5 case series [33-37] 8 case reports [4,38-44]	41 patients	- T2 and FLAIR hyperintensities in the caudate, lenticular nuclei, cerebral cortex, substantia nigra, hippocampus, and internal capsules - In few patients DWI hyperintense white and deep gray matter and splenium of the corpus callosum	Associated with vegetative state or severe disability in two case series
Hyperglycemic encephalopathy			
CT 2 case reports [45,46] 1 case series [47]	11 patients	Hyperdense putamen and/or caudate nucleus or, in fewer patients, normal CT	No clear evidence
MRI 5 case series [47-51] 3 case reports [45,46,52]	31 patients	Uni- or bilateral T1 hyperintensities in the striatum (mostly the putamen)	No clear evidence
SPECT 1 case series [47]	3 patients	Hypoperfusion of 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; PET = positron emission tomography; SPECT = single photon emission computed tomography

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