Supplementary table 1: Characteristics of 4 neurodegenerative disease with possible psychiatric manifestations as a prodrome

		Binswsanger Disease (BD)	**Alzheimer's Disease (AD)**	**Argyrophilic Grain Disease (AGD)**	**Dementia with Lewy Bodies (DLB)**
Prevalence		Variable 1-5 % (1)	First leading cause of dementia (2)	Around 5% (3)	10 % (2)
Risk Factors		HTN, DM (4)	Age; genetics (Apo-e4; APP, PSEN1 and 2); family history of dementia (5)	Age (3)	Genetics, age
Age of onset		≥ 5th decade (1)	65 (5)	8th decade (6, 7)	6th decade (8)
Clinical Presentation	Motor	Extrapyramidal, pyramidal and cerebellar signs, Pseudobulbar signs (4)	Pyramidal and extrapyramidal, myoclonus, seizures (late onset)(5)	-	Parkinsonism, Brady and akinesia, limb rigidity, and gait disorder (8, 9)
	Cognitive	Confusion, impaired judgement, executive dysfunction more prominent than in AD, impaired processing speed, memory dysfunction less common (4)	Specific clinical phenotype of an early and significant episodic memory impairment (10), executive function and judgement impairment (5)	No distinctive clinical or imaging phenotype (11) Mild Cognitive impairment most commonly (11), Amnesia (6)	Fluctuations in cognition and levels of alertness; non amnestic cognitive impairment, (8, 9)
	Psychiatric	Affective disorders (depression mainly), personality changes, late onset paranoid psychosis (1, 12)	Apathy, social disengagement, irritability, aggression, psychosis (5)	Emotional and personality changes, late onset schizophrenia and delusional disorder (11), dysphoria, irritability, agitation, apathy (6), post stroke depression, bipolar disorder (13)	REM sleep behaviors disorder, visual hallucinations, depression, delirium (8, 9, 14)
	Others	Pseudobulbar signs, urinary incontinence (4)	-	-	Orthostatic hypotension, constipation, hyposmia (9)
Diagnosis		Rosenberg criteria: 1- presence of clinical criteria (HTN, DM, hyperreflexia, gait imbalance 2- executive dysfunction on neuropsychological testing 3- Metabolite in WM, 4- Inflammation and Blood Brain Barrier 5- Ruling out AD (15)	Clinical phenotype + One of the following: decreased Aβ1–42 together with increased T-tau or P-tau in CSF, or Increased tracer retention on amyloid PET, or AD autosomal dominant mutation present (10)	Postmortem (16)	Probable DLB: presence of at least one central features (Severe progressive functionally impairing dementia, and deficits on tests of attention/ executive function and visuospatial) + at least one other features either core (fluctuating cognition, recurrent visual hallucinations, spontaneous parkinsonism) or suggestive

				features (REM sleep behaviors disorder, severe sensitivity to antipsychotics, low dopamine transporter uptake in the basal ganglia). (8, 9)
Imaging	Deep and periventricular WM hyperintensities, Prominent frontal and to a lesser extent temporo-parietal atrophy, lacunar infarcts, and subcortical bleeds (4, 17)	Generalized and focal atrophy; Reduced hippocampal volume; Medial temporal lobe atrophy(5)	Unchanged, or mild diffuse or fronto-temporal cortical atrophy NO obvious atrophy of medial temporal lobe (3)	Diffuse pattern of global grey matter atrophy, Loss of parieto-occipital white matter integrity, relatively preserved medial temporal lobe (8)
Pathognomonic Pathological findings	arteriolosclerosis, lipohyalinosis, and fibrinoid necrosis, bystander inflammatory demyelination (15)	Neurofibrillary tangles; senile plaques (18)	Argyrophilic grains (Gallyas positive, Tau positive) Tauopathy? (3)	α-synuclein neuronal inclusions (8)

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