Cases The present case series describes five patients diagnosed with Mills syndrome.

The patient cohort comprises four males and one female, with a mean age at onset of 61 years. Clinical follow up over eleven years established that all patients have disease, most prominently limb weakness, confined to one side of their body. Spasticity was a key feature in all patients, with asymmetric reflexes, brisk on the symptomatic side, and normal sensory examination.

Magnetic resonance (MR) tractography established marked asymmetry of the white matter fiber density, corresponding with clinical presentation. Brain imaging with positron emission tomography (PET) scan identified hypometabolism involving the left lateral frontal cortex posteriorly corresponding to the symptomatic side. Transcranial magnetic stimulation (TMS) showed inexcitability of the motor cortex of the affected side, and motor cortex excitability within normal limits on the contralateral side, consistent with disease confined to one hemisphere.

Discussion Mills syndrome represents a unique phenotype of ALS. Structural imaging, in particular MR tractography, combined with metabolic (PET imaging) and the assessment of cortical motor function promote an accurate diagnosis. Further understanding the pathophysiology of Mills syndrome will broaden understanding of motor neuron disorders, and perhaps expand knowledge of the ALS-PLS spectrum.

3203 ISOLATED BULBAR PALSY VARIANT OF AMYOTROPHIC LATERAL SCLEROSIS (ALS) IN A PATIENT WITH A PATHOGENIC SUPEROXIDE DISMUTASE 1 (SOD1) MUTATION

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Background SOD1 mutations are the second most commonly implicated genetic cause of ALS. Many SOD1 variants have been identified, with few known to be linked to a defined clinical phenotype. 88% of patients with mutations in SOD1 are diagnosed with limb-onset disease. Only 2% of SOD1 patients are diagnosed with bulbar onset disease, which usually confers a significantly poorer prognosis.

Isolated bulbar palsy is a rare variant of ALS, characterised by symptoms confined to the bulbar region for a prolonged period of time, with relative sparing of the limbs. It comparatively confers a significantly better prognosis.

We describe a case of isolated bulbar palsy in a patient with ALS due to a SOD1 mutation (A-G, H43R).

Cases After initial presentation with mild dysarthria and hypophonia, a 36 year-old female was diagnosed with ALS. Her condition remained isolated until 4 years into her disease, when she noticed mild impairment of dexterity in her upper limbs. She remains asymptomatic from a respiratory perspective, and her weight has been stable, without dysphagia since her initial diagnosis with ALS.

At the most recent review 14 years post-diagnosis, there was mild to moderate dysarthria with hypophonia. Limb examination revealed mild weakness of her upper limbs, in a distal pattern.

Discussion ALS is a heterogeneous disease. While ALS represents a universally fatal condition, with a typical lifespan of 2–3 years, rare SOD1 variants may exhibit unexpected disease trajectories, which may confound approaches in the advent of antisense oligonucleotide therapy.

3207 CASE SERIES OF MULTISYSTEM PROTEINOPATHY DUE TO VALOSIN-CONTAINING PROTEIN (VCP) GENE VARIANTS: AN INCONSISTENT PHENOTYPE

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Valosin-containing protein (VCP) gene mutations are an under recognised autosomal dominant genetic disease presenting with both neurological and non-neurological phenotypes. A missense mutation in VCP can cause a myopathy, Paget's disease of the bone or frontal temporal dementia. Less commonly seen phenotypes are amyotrophic lateral sclerosis, parkinsonism or axonal CMT. These conditions are not classically recognised as genetically related by clinicians when taking a family history, so the pattern of autosomal dominance is under recognised. This report will describe a case series with varied clinical presentations, gender, and age where VCP missense pathogenic variants were found. It will also describe a case with a variant of uncertain significance in the VCP gene. Valosin-containing protein is involved in regulatory cellular processes such as autophagy, membrane fusion, transcription and cellular degradation. Most pathogenic missense variants alter the N-terminal ubiquitin binding domains. These missense variants result in failure of regulatory cellular processes, which is thought to account for the wide spectrum of body systems affected. This has led to these diseases to be more recently termed VCP Multisystem Proteinopathies (VCP-MSP). It is pertinent that any family history with a combination of these conditions, including varying severity of symptoms, should raise the question of testing for this condition.

3208 EXPERIENCE AND PRELIMINARY RESULTS OF ESTABLISHING A RESEARCH RECRUITMENT DATABASE FOR FND

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Background/Objectives Functional neurological disorder (FND) is a common, disabling, and usually chronic neurological condition. After a long period of neglect, research interest in the area has grown rapidly in recent years. People with FND (pwFND) often express interest in enrolling in any research studies on the condition, and recruitment is frequently a rate limiting factor in studies.

The traditional method of recruiting for studies: advertising and relying on prospective participants to make contact, is