visual impairment (PSVI). **Methods:** We conducted a quality improvement initiative to create a standardized screening and referral process for patients with PSVI to access an orthoptist. Post-stroke visual impairment was assessed by way of the Visual Screen Assessment (VISA) tool, administered by an occupational therapist. Patients filled out a VFQ-25 questionnaire before and after orthoptic assessment and intervention. The VFQ-25 is a validated post-stroke survey assessing a patient's perceived quality of life. Differences between pre- and post-orthoptic assessment scores will be evaluated. **Results:** Data collection currently ongoing.The benefits of a standardized screen for PSVI, standardized referral to, and experience with an orthoptist assessment will be determined. Learnings gained will also inform how we can expand the program to benefit a wider demographic of patients. **Conclusions:** The data gathered and the subsequent analysis will be instrumental in guiding ongoing improvement initiatives for patients with PSVI.

P.087

Successful response to rituximab in a patient with Aβ related angiitis

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Background: Aβ-related angiitis (AβRA) is a rare presentation of cerebral amyloid angiopathy, where vasculitis results from an auto-immune reaction to amyloid deposits in leptomeningeal and cortical vessel walls. Anti-CD20 monoclonal antibodies, such as rituximab, have demonstrated efficacy in systemic small vessel vasculitides, particularly in refractory cases. The efficacy of rituximab in AβRA remains unknown. **Methods:** Patient chart, functional measures, and laboratory findings were reviewed from the time of patient admission until 12 months after discharge. **Results:** A 61-year-old man presented with headache and altered mental status. Brain MRI revealed multiple cortical infarcts, leptomeningeal enhancement, and cortical microbleeds, and brain biopsy ultimately confirmed the diagnosis of AβRA. The patient developed new ischemic lesions despite corticosteroid pulse, and intravenous cyclophosphamide was halted after four weeks due to iatrogenic acute hepatitis. Rituximab was initiated and led to sustained clinical improvement with no subsequent relapses. Maintenance therapy involved gradually tapered low-dose oral steroids and rituximab at 6- and 12-months post-induction. **Conclusions:** This report suggests that rituximab may be effective in inducing remission and preventing relapses in biopsy-proven case of AβRA. Controlled studies are needed to better assess the efficacy and tolerability of anti-CD20 antibodies in cerebral vasculitis.

P.088

Dysautonomia and Diabetes: A Prodrome to Fatal Familial Insomnia (FFI)

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Background: Fatal Familial Insomnia (FFI) is an autosomal dominant multisystem prion disease, with sleep disorders often being the first presentation. Although autonomic dysfunctions are key features, the frequency and timing vary between reports, and may accompany early insomnia. Moreover, endocrine changes are reported, but diabetes rarely is - with unclear timing of onset in relation to the insomnia. **Methods:** N/A **Results:** Here we present a 46-year-old previously healthy male, who within 22 months prior to the onset of sleep disturbances, developed hypertension and diabetes. Then within 3-4 months after onset of sleep disturbances development tachycardia and diaphoresis. His sleep continued to deteriorate, and later developed bulbar impairment, ataxia, diplopia, sleep apnea and cognitive decline. He passed away 20 months from onset of insomnia. Polysomnography showed status dissociates and central apnea. He had positive genetic testing, PRNP c.532G>A (p.Asp178Asn) and PRNP c.385A>G (pMet129Val), a pathological confirmation, and a positive family history **Conclusions:** Here diabetes and hypertension significantly preceded sleep disturbances, and tachycardia and diaphoresis developed shortly after. This illustrates that dysautonomia and endocrine dysfunction may be unrecognized prodromes in some cases of FFI, and could be an early marker of clinical disease onset and therapeutic interventions, especially in genetically confirmed asymptotic patients.

P.089

A report of a patient presenting with orbital apex syndrome secondary to NK cell lymphoma (nasal type)

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Background: Orbital apex syndrome (OAS) can be caused by a broad range of disorders. There are several challenges present in the evaluation of these patients and in reaching a final diagnosis. We report the case of a 69-year-old male who presented with OAS that was determined to be secondary to a rare malignancy (NK cell lymphoma, nasal type). **Methods:** We analyze the