Methods We reviewed the literature surrounding this rare entity by using PubMed/OVID databases and the search terms ‘Headache AND ecchymosis’. 

Results Case reports exist in older patients1-3, where the headache is side locked and associated with other autonomic characteristics such as periorbital oedema, conjunctival injection and tearing. Our case is a young female with only ecchymosis in a unilateral and/or bilateral manner and no other autonomic or indeed migraine features. The patient underwent vascular/cranial imaging and blood tests to exclude haematological, autoimmune, vasculitic causes for this presentation which were unrewarding.

Conclusion Variations on this clinical entity are described;1-4 we hope this report may bring attention to this fascinating phenomenon. The pathophysiological process is likely to be similar to those implicated in TACs, namely activation of the trigemino-neurovascular system and facial autonomic pathways. The release of neuromediators such as CGRP, VIP and Substance P cause blood vessel fragility resulting in diapedesis. Optimal treatment regimens are unknown but various agents have been trialled. Our patient declined treatment and continues to be followed.

REFERENCES

064 PARKINSON’S DISEASE AND THE GASTROINTESTINAL MICROBIOME: CLINICOPATHOLOGICAL CORRELATIONS AND CONTROVERSIES

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Introduction Parkinson’s disease (PD) is a chronic progressive neurodegenerative disorder characterized by motor symptoms such as tremor, rigidity, postural instability, and bradykinesia. The pathophysiological process is complex and involves multiple pathways and mechanisms, including oxidative stress, inflammation, and immune dysregulation. The gut microbiota has emerged as a potential modulator of PD, as alterations in the gut microbiome have been associated with increased risk of PD, as well as clinical severity and progression.

Methods We conducted a systematic literature review to evaluate the association between Parkinson’s disease and the gut microbiota. We performed a search of PubMed, Ovid, and Scopus databases using relevant keywords such as Parkinson’s disease, gut microbiome, and clinical features. We included studies that investigated the gut microbiome in PD patients compared to healthy controls, as well as those that correlated gut microbiome features with clinical outcomes.

Results A total of 89 studies were identified, of which 43 studies met the inclusion criteria. The majority of studies reported an increase in Firmicutes and a decrease in Bacteroidetes in PD patients compared to healthy controls. Moreover, gut microbiome alterations were associated with disease severity, as measured by the Unified Parkinson’s Disease Rating Scale (UPDRS) and the Hoehn and Yahr stage.

Conclusions Our review highlighted the potential role of the gut microbiome in the pathogenesis of Parkinson’s disease. These findings support further research into gut microbiome modulation as a therapeutic strategy for PD.

REFERENCES

065 PRESENCE OF ANTI-MYELIN OLIGODENDROCYTE GLYCOPROTEIN ANTIBODIES IN THE SERUM OF TWO PATIENTS FOLLOWING ALEMTUZUMAB THERAPY FOR SUSPECTED MULTIPLE SCLEROSIS

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Introduction Myelin oligodendrocyte glycoprotein (MOG) antibody mediated disease is an autoimmune demyelinating disorder which can resemble multiple sclerosis (MS).1 2 Thus, this condition can be misdiagnosed and treated as MS.3 We present the clinical trajectory of two cases initially diagnosed as MS, treated with Alemtuzumab followed by clinical and radiological deterioration. Both were subsequently found to have anti-MOG antibody in their serum.

Methods This is a retrospective case study based on a medical record search of neuroimmunology clinics in two teaching hospitals in Victoria. We searched for patients treated with Alemtuzumab who subsequently tested positive for MOG antibody.

Results We found two young women who fulfilled the eligibility criteria. One patient presented with dizziness and vertigo, the other with unilateral optic neuritis. Both had supratentorial MRI lesions and were both diagnosed as having MS. Both patients experienced multiple relapses while on treatment for MS. Hence, they were commenced on Alemtuzumab therapy. Unexpectedly, both patients experienced a decline in their clinical status with worsening of expanded disability status scale (EDSS) and an increasing lesion load on MRI brain. Their serum anti-MOG antibodies were then found to be positive. Subsequently, patients were treated with rituximab and plasma exchange with a favorable response.

Conclusions These two cases demonstrate that Alemtuzumab is ineffective and in fact can worsen cases of anti-MOG antibody associated encephalomyelitis. This highlights the importance of anti-MOG antibody testing when patients diagnosed with MS do not respond to Alemtuzumab and in those patients presenting with atypical features of MS.

REFERENCES