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

- DeBroff B, et al. Migraine Associated with Periorbital Ecchymosis. *Headache* 1990;**30**:260–263.
- Dafer R, et al. Atypical Chronic Headache and Recurrent Facial Ecchymosis: A Case Report. *Neuro-Ophthalmology* 2011;**35**:76–77.
- Nozzolillo D, et al. Migraine associated with facial ecchymoses ipsilateral to the symptomatic side. *J Headache Pain* (2004) **5**:256–259.
- Sethi PK, et al. Teaching neuroimages: Red forehead dot syndrome and migraine revisited. *Neurology* 2015;**85**:e28.

064

PARKINSON'S DISEASE AND THE GASTROINTESTINAL MICROBIOME: CLINICOPATHOLOGICAL CORRELATIONS AND CONTROVERSIES

¹Michal Lubomski*, ²Ai Huey Tan, ²Shen-Yang Lim, ³Andrew Holmes, ¹Ryan L Davis, ¹Carolyn M Sue. ¹Department of Neurogenetics, Kolling Institute, University of Sydney and Royal North Shore Hospital, Sydney, NSW, Australia; ²Neurology Department, Mah Pooi Soo and Tan Chin Nam Centre for Parkinson's and Related Disorders, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia; ³School of Life and Environmental Sciences, The Charles Perkins Centre, University of Sydney, Sydney, NSW, Australia

10.1136/jnnp-2019-anzan.57

Introduction There has been a recent surge in interest around the gastrointestinal microbiome (GM) and its association with Parkinson's disease (PD). The GM mediates interactions between the brain and the gut via the 'microbiota-gut-brain-axis'. Compelling studies suggest that a shift in GM composition may play an important role in the pathogenesis and progression of PD.

Methods We conducted a literature review exploring the pathological association between the GM, α -synuclein spread and intestinal inflammation in PD. We also summarised patterns and correlations of gut microflora seen in clinical studies of the GM in PD.

Results To date 14 mainly cross-sectional studies from 7 countries have reported GM alterations in PD. All studies described significant alterations between PD and healthy control groups across multiple bacterial families, genera and

species. Several studies suggested that putative 'pro-inflammatory' bacteria were significantly more abundant, while putative beneficial bacteria were less abundant in PD. Various complex microbiota-gut-brain-axis interactions have been proposed due to alterations in the GM, inferred by changes in gut mucosal integrity and permeability, short-chain-fatty-acid metabolism, oxidative stress and inflammation.

Conclusions Across the recent GM studies in PD, alterations in bacterial taxa have been repeatedly associated with various clinicopathological features, endorsing a plausible biological link between the GM and PD. Mechanisms involved in the pathogenesis of PD due to GM changes are complex and require ongoing study.

065

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

^{2,1}Sinali Seneviratne*, ²Mark Marriott, ²Mastura Monif. ¹Medical School, Curtin University, Perth, WA, Australia; ²Department of Neurology, Royal Melbourne Hospital, Melbourne, VIC, Australia

10.1136/jnnp-2019-anzan.57

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

- Weber MS, Derfuss T, Metz I, Bruck W. Defining distinct features of anti-MOG antibody associated central nervous system demyelination. *Ther Adv Neurol Disord.* 2018;**11**:1756286418762083.