Background Autoimmune encephalitis is an increasingly recognised disease that presents with seizures, neuropsychiatric symptoms, dystonic movements, and autonomic dysfunction[1]. As the mainstay of treatment immunosuppressive therapies are most effective when subject to early initiation and timely escalation, both of which are recognised to affect outcomes[2]. Nevertheless approximately half of patients with NMDA-R antibody encephalitis do not respond adequately to first-line therapy, and a significant proportion (12-30%) relapse[2].

Cases A 19 year old lady presented with new-onset seizures and psychosis. EEG showed focal spike-and-wave discharges and MRI brain a focal area of restricted diffusion consistent with recent seizure activity. NMDA-R antibodies were present in both CSF and serum. Following early treatment with corticosteroid, plasma exchange, IVIG and rituximab the patient recovered, returning to college after 6 months.

A 50 year old gentleman presented with a two day history of myalgias and confusion. EEG showed spike-and-wave discharges and MRI brain increased T2 signal in the mesial temporal lobes. NMDA-R antibodies were present in both CSF and serum. He was treated with corticosteroid, plasma exchange, IVIG and rituximab, and continued on oral prednisone and mycophenolate. Response to treatment was poor with persistent ongoing physical and cognitive impairment at 6 months. Serial MRI showed substantial (~30%) loss of parenchymal brain volume.

Discussion These cases illustrate that timely and aggressive management of NMDA-R antibody encephalitis with favourable prognostic markers is no guarantee of recovery. Several novel clinical and immunological predictors of response to therapy have been postulated, and currently await broader validation.[3]

REFERENCES

1. Dalmau J, Graus F. Antibody-mediated encephalitis. *N Engl J Med* 2018 Mar 1;**378**(9):840–851.
2. Titulaer MJ, McCracken L, Gabilondo I, Armangué T, Glaser C, Iizuka T, *et al*. Treatment and prognostic factors for long-term outcome in patients with anti-NMDA receptor encephalitis: an observational cohort study. *Lancet Neurol* 2013;**12**(2):157–65.
3. Rüegg S, Yeh EA, Honnorat J. Forecasting outcomes in anti-NMDAR encephalitis: clearer prognostic markers needed. *Neurology* 2019 Jan 15;**92**(3):119–120.

108 THE DIAGNOSIS AND MANAGEMENT OF FIVE CASES OF SPINAL NEUROSARCOIDOSIS

[1]Ariadna Fontes-Villalba, [2]Dayna Griffiths, [3]Natasha Gerbis, [1]Jonathan Baird-Gunning, [4]Patrick Aouad, [5]Suran Fernando, [1]John Parratt. *[1]Neurology, Royal North Shore Hospital, Sydney, NSW, Australia*; *[2]Neurology, Neurology Dept. Gosford Hospital, Gosford, NSW, Australia*; *[3]Neurology, Northern Beaches Hospital, Frenchs Forest, NSW, Australia*; *[4]Department of Neurology, Liverpool Hospital, Sydney, NSW, Australia*; *[5]Department of Immunology, Royal North Shore Hospital, Sydney, NSW, Australia*

10.1136/bmjno-2021-ANZAN.108

Objectives Neurosarcoidosis may present with longitudinally extensive transverse myelitis (LETM) posing a diagnostic challenge. We describe the clinical features, radiology and management of five patients with spinal neurosarcoidosis (SNS).

Methods We retrospectively identified five patients with a diagnosis of SNS and the clinical, radiological and pathological data were reviewed.

Results There were three females and two males who were, on average, 49 years at onset. A histopathological diagnosis of sarcoid was confirmed in two cases (lymph node and cerebral lesion) and the diagnosis was radiological in the others (LETM with persistent enhancement in 3 patients and the trident sign in 2 patients). The average span of the spinal cord lesions was 4 vertebral bodies. Three patients had FDG-avid mediastinal lymph nodes. Cerebral disease was identified in two cases, and cardiac involvement in one. Two patients required spinal decompression surgery. All patients received intravenous and oral steroids and some had rituximab (n=2), tocilizumab (n=1), cyclophosphamide(n=1), adalimumab (n=2), and infliximab (n=4). Disease control was achieved with TNF-alpha (tumour necrosis factor-alpha) blocking in 4 cases and another responded to cyclophosphamide. The mean follow-up was 55 months.

Conclusions SNS is a cause of LETM and can be suspected by the trident sign on MRI. Persistent enhancement may be another differentiating feature. Spinal cord oedema requiring surgery may occur and patients typically respond to treatment with TNF-alpha inhibitors.

REFERENCES

1. Flanagan EP, Kaufmann TJ, Krecke KN, Aksamit AJ, Pittock SJ, Keegan BM, Giannini C, Weinshenker BM. Discriminating long myelitis of neuromyelitis optica from sarcoidosis. *Ann Neurol* 2016 Mar;**79**(3):437–47.
2. Zalewski NL, MD, Krecke KN, Weinshenker BG, MD, Aksamit AJ, Conway BL, McKeon A, Flanagan EP. Central canal enhancement and the trident sign in spinal cord sarcoidosis. *Neurology* 2016 Aug 16;**87**(7):743–4.

109 PAIR-WISE DIFFERENCES OF PENUMBRA AND CORE VOLUME ESTIMATES FROM THREE COMPUTED TOMOGRAPHY PERFUSION SOFTWARE PACKAGES ARE INFLUENCED BY SITE OF LARGE VESSEL OCCLUSION

[1,2]Peter SW Park, [1]Robbie Chan, [1]Channa Senanayake, [1]Stanley MK Tsui, [2]Alun Pope, [1,2]Helen M Dewey, [1,2]Philip MC Choi. *[1]Department of Neurosciences, Box Hill Hospital, Eastern Health, Box Hill, VIC, Australia*; *[2]Eastern Health Clinical School, Faculty of Medicine, Nursing and Health Sciences, Box Hill, VIC, Australia*

10.1136/bmjno-2021-ANZAN.109

Objectives Computed tomography perfusion (CTP) data are important for hyperacute stroke decision making. Comparisons between outputs of different CTP software packages are limited. We aimed to assess the pair-wise differences in infarct and penumbra estimates produced by three CTP software packages – MIStar, RAPID, and Vitrea.

Methods Consecutive patients with suspected acute ischaemic stroke who underwent CTP between July 2020 and June 2021 at our hospital were independently reviewed by two expert readers. Pair-wise differences between software estimates of penumbra and core volumes were calculated for each patient, with analysis stratified by large vessel occlusion (LVO) status (no-LVO, proximal M2, M1 and internal carotid artery-T [ICA-T]).

Results 580 CTP studies were performed; 262 were normal, 146 technically poor, with 172 included in the final analysis. 79/172 (45.9%) had LVO; proximal M2 (n=21), M1 (n=38) and ICA-T (n=20). Overall, statistically significant pair-wise differences were seen for both penumbra and core estimates ($P < 0.001$). The largest difference in mean core estimates were seen between Vitrea and MIStar ([mean, 95% confidence interval] no-LVO [5.8ml, 3.2–8.4]; proximal M2 [10.4ml, 3.9–17.0]; M1 [17.7ml, 8.9–26.6]; ICA-T [38.9ml, 20.2–57.7]). More comparable penumbra estimates were observed between RAPID and MIStar (no-LVO [1.79ml, -3.9–7.51]; proximal M2 [13.1ml, -0.24–26.5]; M1 [10.7ml, -5.9–27.3]; ICA-T [28.4ml, 0.78–56.0]).