

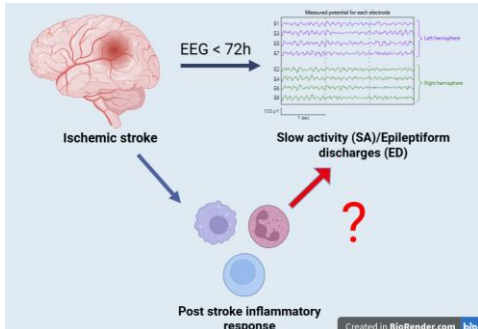
THE ROLE OF POST STROKE INFLAMMATION IN ACUTE EEG ALTERATION

Gabriele Prandin¹ MD, Giovanni Furlanis¹ MD, Laura Mancinelli² MD, Federica Palacino¹ MD, Emanuele Vincis¹ MD, Edoardo Ricci¹ MD, Magda Quagliotto¹ MD, Michele Malesani¹ MD, Paola Caruso¹ MD, Giulia Mazzon¹ MD, Marinella Tomaselli¹ MD, Marcello Naccarato¹ MD PhD, Paolo Manganotti¹ MD PhD

Clinical Unit of Neurology, Department of Medicine, Surgery and Health Sciences, ASUGI, University of Trieste, Trieste, Italy

Background and aims

Post stroke inflammation is a well-known complication which plays a significant role in outcome prediction. Previous studies found a strong association of both slow wave activity (SA) and epileptiform discharges (ED) with stroke outcome. However, there are few data about the relationship between inflammation and acute phase EEG alteration.



Materials and methods

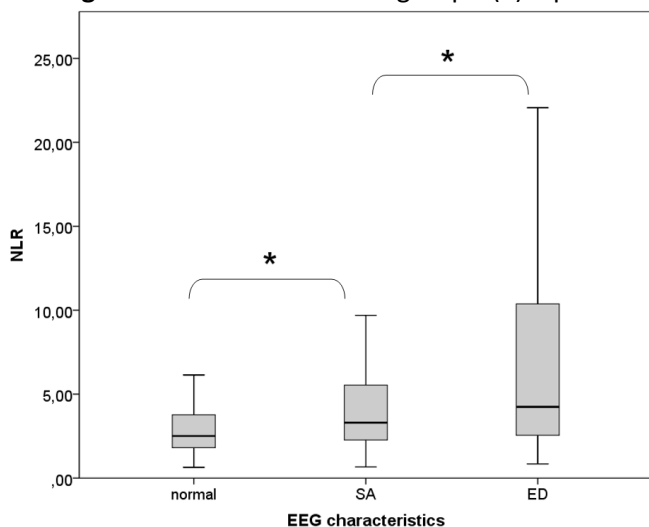
We retrospectively analysed data of patients with an EEG recording after admission to the SU of Trieste. We excluded haemorrhagic strokes, stroke mimics, infratentorial ischemic strokes, factors which can alter the inflammatory biomarkers (infections, immunosuppressant drugs) and factors which can modify the EEG (previous strokes, antiseizure medication, cognitive impairment). The blood tests were collected after 24h the admission (e.g., C-reactive-protein (CRP), erythrocyte sedimentation rate (ESR), white blood count, neutrophil and lymphocyte count and their ratio (NLR)). We compared the stroke characteristics and risk factors between SA vs no-SA and ED vs no-ED. Then we performed a multivariate analysis (logistic regression).

Results

316 patients were analysed. 140 have SA and 67 ED with/without SA. After multiple adjustment, NLR was not associated with SA (OR 0.98, CI95% 0.89-1.08, $p=0.709$), however NLR (OR 1.01, CI95% 1.03-1.16, $p=0.002$) remains significantly associated with ED. SA remains associated with some stroke characteristics as admission NIHSS (OR 1.15, CI95% 1.07-1.24, $p<0.001$) and haemorrhagic transformation (OR 4.23, CI95% 1.26-14.21, $p=0.020$).

MULTIVARIATE Regression for SA		
	OR (95% CI)	p
Treatment	1.229 (0.667-2.264)	0.509
AF	1.140 (0.415-3.131)	0.800
OCSF		0.144
TACI	4.483 (0.823-24.413)	0.083
PACI	1.412 (0.397-5.016)	0.594
LACI	0.981 (0.889-1.083)	0.709
POCI	Ref	
HT	4.233 (1.261-14.207)	0.020
Admission NIHSS	1.152 (1.068-1.242)	<0.001
NLR	0.981 (0.889-1.083)	0.709
MULTIVARIATE Regression for ED		
	OR (95% CI)	p
Female sex	2.245 (1.203-4.191)	0.011
Treatment	1.210 (0.635-2.305)	0.562
AF	1.032 (0.532-2.001)	0.926
OCSF		0.102
TACI	1.362 (0.347-5.343)	0.657
PACI	0.516 (0.150-1.771)	0.293
LACI	0.577 (0.153-2.183)	0.418
POCI	Ref	
Admission NIHSS	1.014 (0.961-1.071)	0.602
NLR	1.097 (1.033-1.164)	0.002

Figure 1: NLR levels in the 3 groups. (*) if $p<0.01$



Conclusions: Post stroke inflammation may play different roles in acute EEG. NLR may be more related to the development of ED than SA. SA on EEG is more related to stroke severity and haemorrhagic transformation.