# MECHANISMS OF DAMAGE IN ACUTE STROKE

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## Introduction

Monocyte Distribution Width (MDW) reflects peripheral monocyte activation in the context of innate immune response against pathogenic noxae. It is an early marker of sepsis. The aim of our study was to assess this parameter in acute ischaemic stroke (AIS).

## Materials and Methods

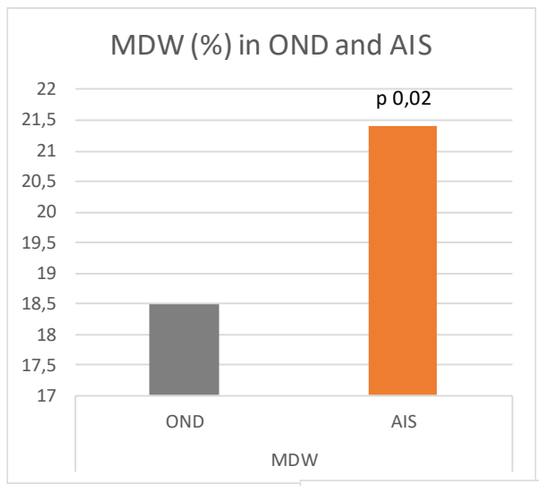
So far, we recruited 24 AIS (age 72 sd 13), 16 Transient Ischaemic Attacks (TIA, age 66,7 sd 14,8), 11 Other Neurological Diseases (OND, 40,1 sd 16,8). They underwent blood withdrawal within 24 hours, 24 hours Arterial Pressure Measurement, Magnetic Resonance Imaging within one week. MDW was detected by Volume, Conductivity, and Scatter technology by DXH900 Ematology Analyzer Beckman Coulter. Lesional load, related to Periventricular White Matter (PWM), Deep White Matter (DWM) lesions was calculated according to Fazekas' criteria. Moreover, we considered Juxtacortical ischaemic lesions (JC) and microbleeds (MB), evaluated as rare (1:*1-3*), sporadic (2:*3-6*), multiple (3:>*6*) at FLAIR-T2 and SWI-Weighted-MRI sequences.

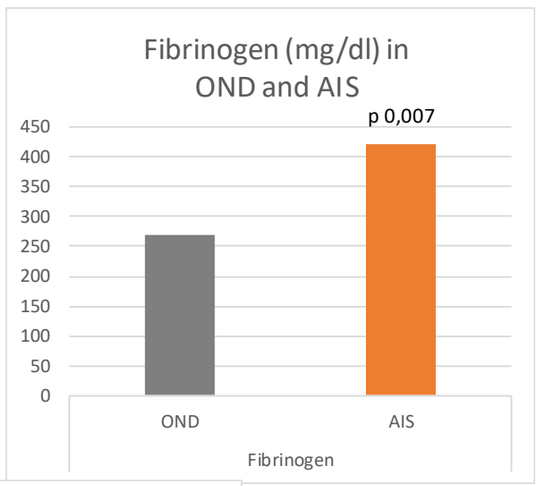
## Results

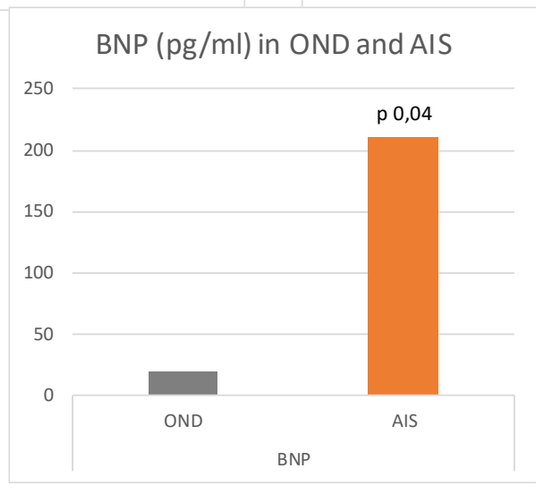
The following parameters were significantly higher in AIS compared to OND: MDW (21,4 sd 3,8 vs 18,5 sd 1,7 %, p 0,02), fibrinogen (421,3 sd 156 vs 269,9 sd 32,6 mg/dl, p 0,007), Brain Natriuretic Peptide (BNP, 210,5 sd 296,5 vs 19,6 sd 16,3 pg/ml, p 0,04), PWM (1,12 sd 1,1 vs 0,17 sd 0,41, p 0,05), DWM (1,27 sd 0,97 vs 0,25 sd 0,4, p 0,02), JC (1,5 sd 0,91 vs 0,42 sd 0,49, p 0,01). Common denominator in TIA and AIS compared to OND was higher Mean Blood Pressure (MBP, 88,7 sd 8,5, p 0,02, 86,5 sd 6,1, p 0,02, vs 79,3 sd 6,4 mmHg).

MDW positively correlates with fibrinogen (r 0,74).

No significant correlation was found with chronic lesional load.




## Discussion

Our data suggest that increased coagulability is the primum movens of the ischaemic cascade, leading to reduced flow and endothelial damage of the Virchow's triad. MDW may be an early marker of the quartet, namely inflammation. Fibrinogen is the precursor of fibrin, entrapping blood cells, among which monocytes. They shift to the activated amoeboid form and produce inflammatory mediators, as interleukins-6 and tumor necrosis factor. They further aggregate platelets, worsening thrombosis. Moreover, they infiltrate the lesion site within 24 hours, reaching peak levels at day 3 after acute event. Lastly, they are antigen presenting cells and may promote autoimmune response. However, our preliminary results on negative correlation with chronic lesional load suggest to focus on acute ischaemic lesion, where resident microglia, activated within minutes to few hours, may be the main culprit of irreversible tissue damage, followed by clearance of cellular debris promoting repair and remodeling. We further highlight earlier the thrombolysis, easier the recanalization and safer the reperfusion. Nonetheless, late window paradox may represent the lag of time before in situ acute, early inflammatory response.

**References**

Juehui Wu, Laisheng Li, Jinmei Luo - Diagnostic and Prognostic Value of Monocyte Distribution Width in Sepsis - Journal of Inflammation Research - 2022 - 15 - 4107-4117