



Pathogenesis of delayed cerebral ischemia in aneurysmal subarachnoid haemorrhage—a multimodality monitoring study

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INTRODUCTION

The aneurysmal subarachnoid hemorrhage (aSAH) is a devastating form of a stroke. One of the most common complication after aSAH is the delayed cerebral ischemia (DCI), defined as a focal or global neurological deterioration, cerebral infarction or both. The cerebral vasospasm has been suggested for a long time as the main reason of DCI, however its contribution in DCI remains unclear. In order to study the DCI pathogenesis and determine the factors, which lead to cerebral ischemia, we used a multimodal continuous monitoring of multiple variables. We aim to study the changes in cerebral autoregulation (CA) and the alterations in the systemic hemodynamic during the early post-hemorrhage period to examine the pathological mechanisms underlying DCI and determine the effect of those changes on the short-term outcome and mortality in aSAH patients.

MATERIALS AND METHODS

A total of 57 patients with a mean age of 56±18 years with diagnosed moderate aSAH (H-H grade 3±1) and hospitalized in Wrocław University Hospital between 2012 and 2017 were prospectively enrolled in the study.

Continuous recording of following physiological signals were conducted using computer-based system Intensive Care Monitor (ICM+):

arterial blood pressure (ABP), intracranial pressure (ICP), end-tidal CO₂ (EtCO₂), regional cerebral oxygenation (rSO₂), cardiac output (CO), cardiac index (CI), stroke volume variation (SVV), stroke volume (SV), stroke volume index (SVI), cerebral blood flow velocity in the middle cerebral artery (CBFV).

Cerebral autoregulation was assessed by applying:

- pressure reactivity index (PRx), tissue oxygenation index (TOx), mean velocity index (Mx).

RESULTS

- 14 patients (25%) died during hospitalization. 74% (n=42) of the survivors had good short-term outcome, whereas 26% (n=15) had poor short-term outcome.
- DCI was seen in 21 patients (37%) while TCD diagnosed vasospasm was seen in 22 patients (39%), which occurs in 5±2 day after onset.
- NIRS measurements were performed in 35 patients. In those group regional cerebral desaturation was found in 17 patients (48%). DCI was found in 37% of patients with cerebral desaturation episodes and in 35% of patients without them.
- Neurophysical impairment after aSAH occurred in 27 patients (47%) and cardiovascular disorders were found in 26 patients (46%).
- The systemic cardiovascular disorders during acute phase have been observed. The CO and SVV were stabilized due to apply a monitored therapy.
- The PRx on 2nd day was significantly higher than on 6th day after onset (0.167 vs. -0.054, p=0.04).
- The CO, CI and SV median values were similar in DCI and non-DCI group: CO [l/min]: 5.3±1.7 vs. 5.1±2.1, CI [l/min/m²]: 2.9±0.9 vs. 3.1±0.9, SV [ml]: 72.8±28.3 vs. 67.0±34.0.

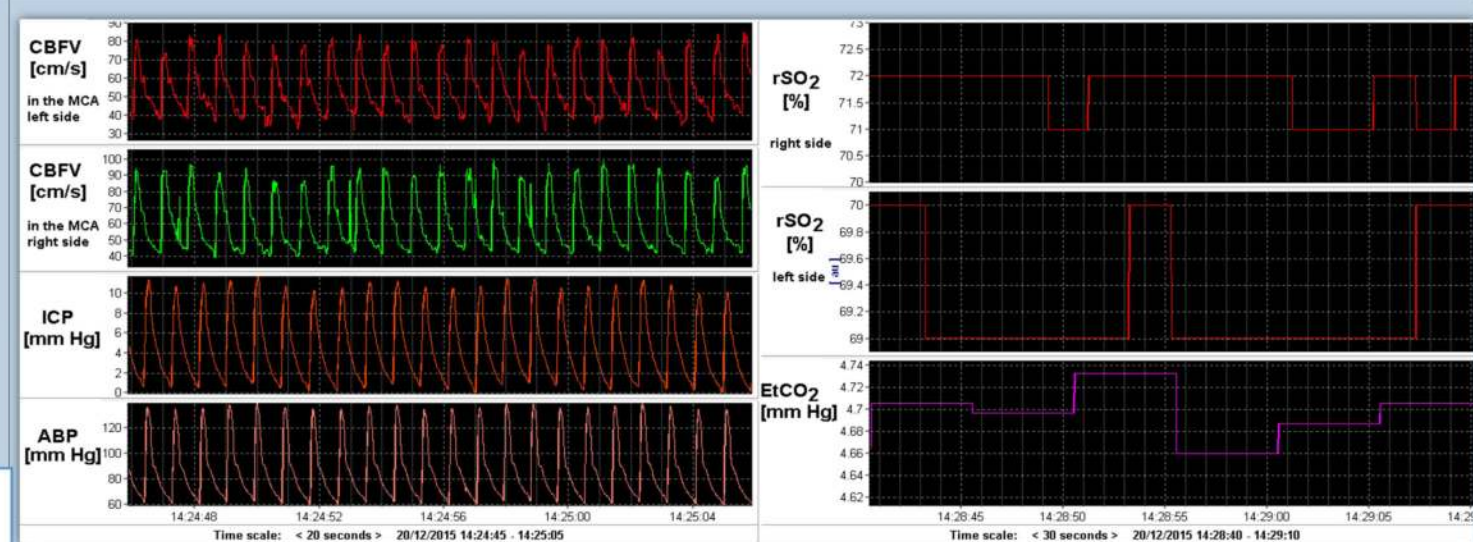


Fig. 1 Example of multimodal monitoring of a 60-year-old man 1 day after aneurysmal subarachnoid haemorrhage from ruptured aneurysm in basilar artery (BA).

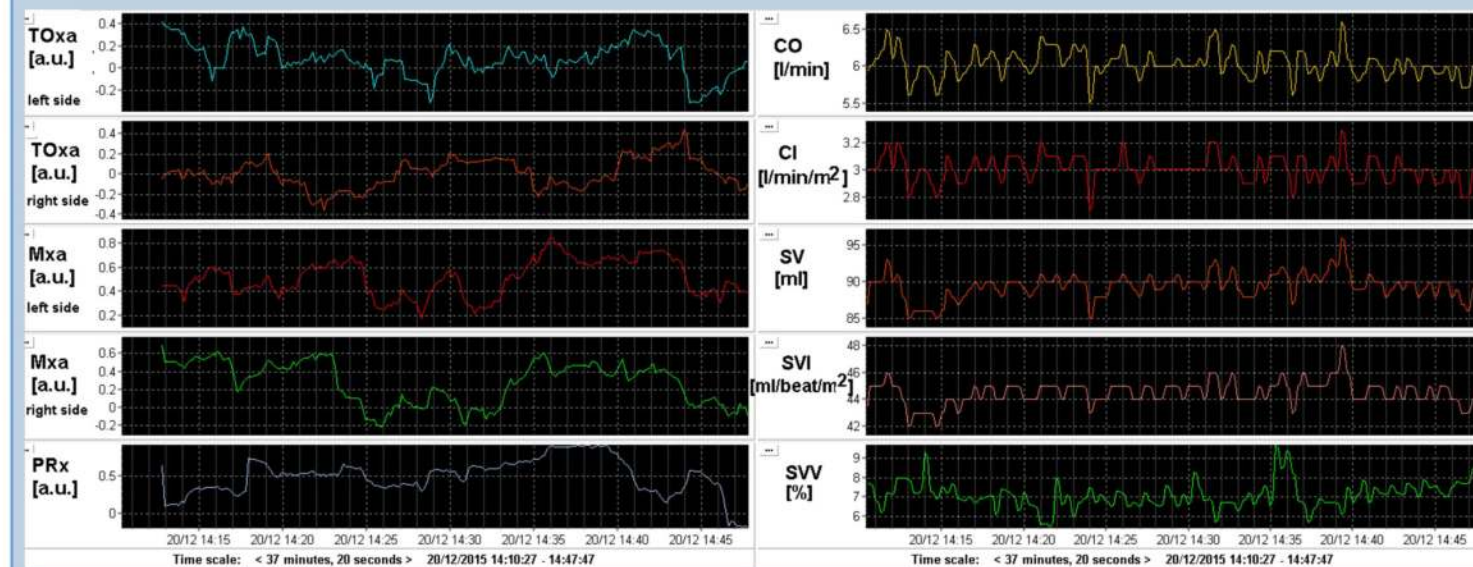


Fig. 2 Example of cerebral autoregulation assessment in real-time mode in a 60-year-old man 1 day after aneurysmal subarachnoid haemorrhage from ruptured aneurysm in basilar artery (BA).

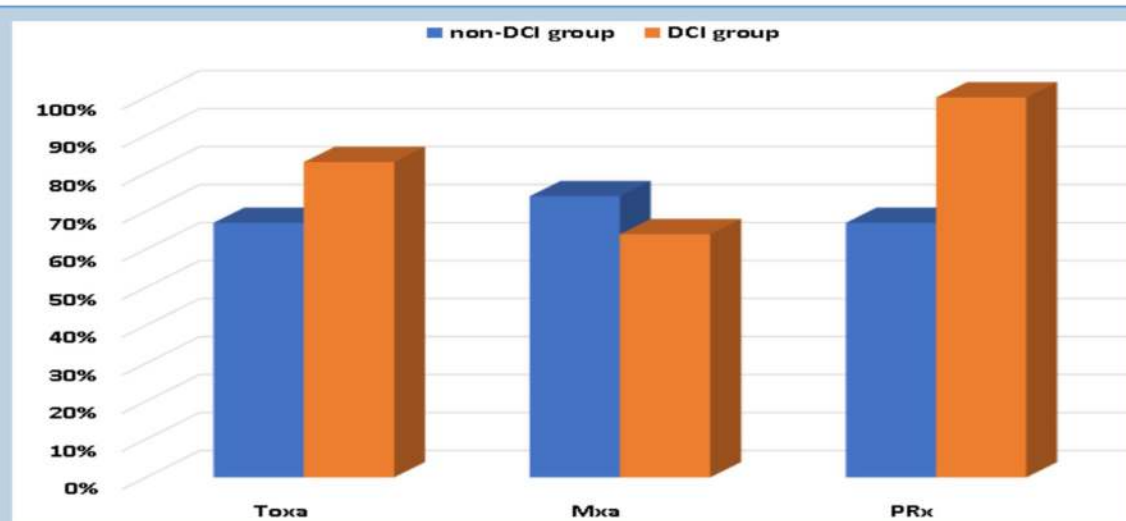


Fig. 3 Pathological values of cerebral autoregulation occurring in DCI and non-DCI group of aSAH patients. Note, that the TOxa, Mxa and PRxa were not determined in entire group: TOxa in 36 patients, Mxa in 41 patients, PRxa in 20 patients.

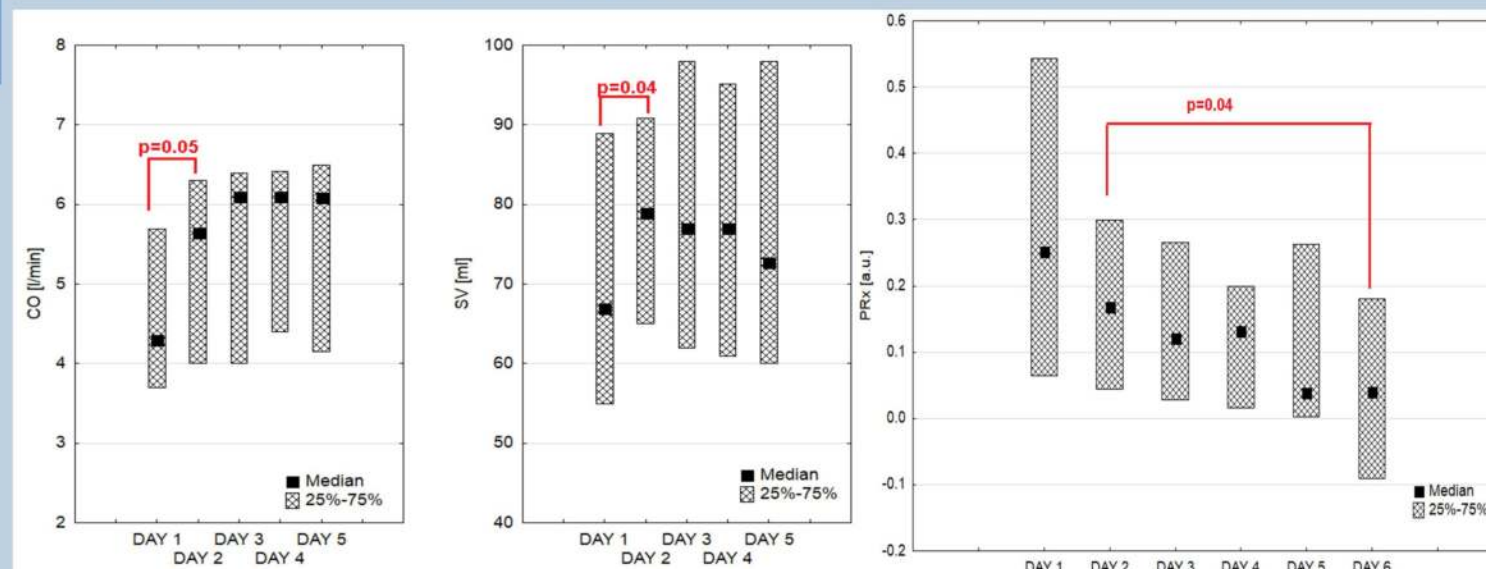


Fig. 4 The median values of cardiac output (CO), stroke volume (SV) and pressure reactivity index (PRx) during following days after onset in total group of aSAH patients.

CONCLUSIONS

The monitored-guided agents therapy, combined with a multimodality monitoring of cerebral autoregulation, autonomic nervous system and cardiovascular system, allows to personalize treatment and may lead to better outcome in aSAH patients. Cardiovascular and pulmonary complications after aSAH were commonly observed in aSAH patients. Thus, standard monitoring should be extended to systemic and cerebral hemodynamic monitoring, despite the absence of cardiac disorders at admission to the hospital.

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