

Cerebral blood flow examination in patients with sepsis associated encephalopathy

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Previous clinical observations have shown that the brain is often the first organ affected by sepsis, preceding the clinical symptoms of other organ manifestations. The pathophysiology of sepsis-associated encephalopathy (SAE) is not entirely clear, but one of the possible underlying mechanisms is the alteration of the cerebral microvascular function. The aim of the present work was to test whether: 1) cerebral vasomotor reactivity is impaired in patients with SAE; 2) cerebral vasomotor reactivity is impaired in patients suffering from severe sepsis.

In the first scenario patients (n=14) fulfilling the criteria of clinical sepsis and showing disturbance of consciousness of any severity were included. In the second scenario patients (n=16) also fulfilling the criteria of clinical sepsis, but showing at least two organ manifestations were included. Non-septic persons without previous diseases affecting cerebral vasoreactivity served as controls in both scenario (n=20; n=16). Transcranial Doppler blood flow velocities were measured at rest and 5, 10, 15 and 20 minutes after intravenous administration of 15mg/kgBW acetazolamide. The time course of the drug effect on cerebral blood flow velocity (cerebrovascular reactivity, CVR) and the maximal vasodilatory effect of acetazolamide (cerebrovascular reserve capacity, CRC) were compared among the groups both cases.

Absolute blood flow velocities after administration of the vasodilator drug were higher among control subjects than in SAE, however in case of second scenario the absolute blood flow velocities did not differ between control and severe septic patients. When assessing the maximal vasodilatory ability of the cerebral arterioles to acetazolamide during vasomotor testing, in the first scenario we found that patients with SAE reacted lesser extent to the drug than control subjects, but in second scenario the severe septic patients reacted to a similar extent than the controls (1. CRC controls: $46.2 \pm 15.9\%$, CRC SAE: $31.5 \pm 15.8\%$, $p < 0.01$; 2. CRC controls: $54.8 \pm 11.1\%$, CRC severe septic: $61.1 \pm 34.4\%$, $p = 0.49$).

We conclude that cerebrovascular reactivity is impaired in SAE, but is not impaired in patients with severe sepsis. Our results indicate that cerebral vasoreactivity may be differently involved at different severity stages of the septic process.

Keywords: Sepsis, Sepsis Associated Encephalopathy, Cerebral Vasoreactivity

Kulcsszavak: Szepszis, Szepsziszhez társuló enkefalopátia, Agyi vazoreaktivitás