

# WHY CORTICOSTEROIDS ARE NOT RECOMMENDED IN STROKE MANAGEMENT

Ivan Bielen

University Hospital "Sveti Duh", Zagreb  
Clinical Department of Neurology

Corticosteroids are very efficient in the management of vasogenic oedema caused by cerebral tumors or CNS infections, but according to the current evidence-based clinical practice guidelines (1-2), corticosteroids are not recommended in standard stroke management, even though the stroke lesions are also often accompanied by formation of vasogenic oedema. Corticosteroid therapy in stroke management was generally discarded after the trials which were made in the last quarter of the 20th century. Based on the meta-analysis from 8 randomised controlled trials that included 466 patients, it was suggested that corticosteroids do not provide evidence of a beneficial effect on death following acute ischemic stroke, and that the obtained results do not support the routine use of corticosteroids in the management of acute ischemic stroke. It was emphasized that, given the possible adverse effects of corticosteroids, the benefits on both the case fatality and functional outcome would need to be substantial for this treatment to be recommended (3). The main adverse effects of steroid therapy that may set off potential positive effects in ischemic cascade or oedema formation are hyperglycaemia, infections, and gastrointestinal haemorrhage.

Although there is no evidence in favour of the use of corticosteroids in stroke management, there are opinions that the clinical investigations had been prematurely discarded. In primary intracerebral haemorrhage (ICH), which is characterised by perihematoma oedema formation, there appear to be only 3 randomized control trials targeting ICH alone, involving only 159 patients (4). The criticism toward the current statements is based on the relatively small number of patients included in the performed clinical trials, unequal methods and conclusions of the analysed trials, but also on the results of some animal studies which showed a beneficial effect of corticosteroids in some experimental stroke models

(4-5). On the other hand, the preclinical studies demonstrated also some potential harmful effects of corticosteroids, e.g. decreased inactivation of glutamate, decreased expression of BDGF and increased neuronal sensitivity on hypoxia (6). In addition to well known clinical undesirable side-effects of corticosteroids, these biochemical changes might counteract the expected positive effects of steroids.

Despite the suggestions that renewed corticosteroid trials in stroke should be designed, it seems that further large-scale trials so far cannot be expected.

## Most relevant references

1. European Stroke Organisation (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis.* 2008;25(5):457-507
2. Demarin V, Lovrenčić Huzjan A, Trkanjec Z, et al.; Croatian Society for Neurovascular Disorders; Croatian Stroke Society; Reference Center for Neurovascular Disorders of Croatian Ministry of Health; University Department of Neurology, Sestre milosrdnice University Hospital. Recommendations for stroke management. *Acta Clin Croat* 2006; 45:219-85
3. Sandercock PAG, Soane T. Corticosteroids for acute ischaemic stroke. *Cochrane Database Syst Rev.* 2011 Sep 7;(9):CD000064
4. Norris JW. Steroids may have a role in stroke therapy. *Stroke.* 2004; 35(1):228-9.
5. Davis SM, Donnan GA. Steroids for stroke: another potential therapy discarded prematurely? *Stroke.* 2004;35(1):230-1
6. Jonathan KJ, Rhodes MB. Actions of glucocorticoids and related molecules after traumatic brain injury. *Curr Opin Crit Care* 2003; 9:86-91