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Letter to the Editor

Corticosteroid-induced myopathy in spinal cord injury patients: a role for anticatabolic agents?

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We read with interest the article by Qian *et al*[1] on 'High-dose methylprednisolone may cause myopathy in acute spinal cord injury patients.' The authors presented interesting data on EMG and muscle biopsies, which demonstrated acute myopathy in patients after receiving methylprednisolone for spinal cord injury. Conflicts over the use of methylprednisolone for spinal cord injury concern the risk-to-benefit ratio.[2,3] A study by the co-workers of Qian *et al* demonstrated that methylprednisolone administered to patients with spinal cord injury upon admission and during the operative procedure significantly increased risk for complications as compared to a single dose upon admission.[4] Corticosteroids are routinely utilized in neurosurgery patients for their inherent ability to decrease edema.[5] Corticosteroids have been shown to have a multitude of deleterious effects on other organ systems.[2–3] A few cases of catastrophic immunosuppression have been reported with routine doses of corticosteroids in the postoperative period.[2,3,6] We have previously reported on the use of Oxandrin (BTG Pharmaceuticals, NJ, USA), which is indicated to combat the deleterious effects of corticosteroids.[7,8] Oxandrin has anticatabolic and anabolic properties by binding to the androgen receptor and initiating an anabolic cascade of tissue building and repair and it is anticatabolic through the weak competitive binding to the corticosteroid receptor thus buffering the catabolic cascade.[9] One might question, would the Oxandrin inhibit the beneficial effects on cerebral and spinal cord edema, and through our own clinical experience it has not been an issue.

Of interest is the recent evidence suggesting that progesterone may be an alternative to corticosteroids in spinal cord injury and traumatic brain injury.[10,11] Progesterone was found to have antiapoptotic and antiastrogliotic effects on the brain and led to improved cognitive performance as compared to control animals with traumatic brain injury.[10] In addition, progesterone has shown to be neuroprotective in spinal cord injury in animals.[11] Are the effects of progesterone solely through the progesterone receptor or is it also binding to the androgen receptor? Progesterone has an affinity to the androgen receptor and thus the actual benefits may be, in part, through the androgen receptor.[9] We commend Qian *et al* on their fine work and propose to the authors to add a third group to the study which would incorporate Oxandrin and methylprednisolone to determine if the anabolic/anticatabolic effects of Oxandrin may prevent myopathy from occurring.

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