Role of nimodipine in severe diffuse head injury

Sir,

I read with interest the article published by Pillai S et al., which is a double blind placebo-controlled trial, evaluating the role of nimodipine in severe diffuse head injury. Although pathological increases in intracellular calcium have been implicated as the major final common pathway leading to neuronal death, the mechanism of increase in intracellular calcium in neurons is largely due to excitotoxicity, following hypoxic-ischemic injury or head trauma. After a severe diffuse head injury, the diffuse neural injury that results is caused primarily by the presence of excess glutamate, due to its action on N-methyl-D-aspartate (NMDA) receptors. The activated NMDA receptor-channels allow an influx of Ca$^{2+}$, which in excess can activate a variety of potentially destructive processes. Nimodipine, because of its high lipid solubility, was developed as an agent to relax cerebral vasculature, and is effective in inhibiting cerebral vasospasm, but does not have any action on NMDA receptors. Hence nimodipine was found to be effective in conditions causing cerebral vasospasm, such as in severe head injury with contusions and intracranial hematomas, where in, its ability to inhibit vasospasm has a significant beneficial effect in reducing neural damage. In this study by Pillai S et al., the patients included showed radiological evidence of only diffuse head injury without any operable mass lesion like intracerebral hematoma or contusion more than 1 cm in diameter, or extradural and acute subdural hematomas more than 1 cm in maximum thickness. It would have been interesting if cerebral vasospasm was demonstrated in these cases with the help of transcranial doppler. This would have provided a better insight into the role of nimodipine in severe diffuse head injury. In such cases with diffuse head injury, excitotoxic injury by glutamate is more likely to be the major cause of neural injury, compared to cerebral vasospasm, as substantiated in this study with no significant improvement in outcome in patients treated with nimodipine, compared to the placebo group. In these patients with diffuse head injury, agents which block NMDA receptors, such as Mg$^{2+}$, may have a beneficial effect.

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Ventriculo-peritoneal shunt infection by mycobacterium fortuitum in an adult

Sir,

A 60-year-old male patient with unconsciousness after an assault was operated for decompression of fracture, and right-sided ventriculo-peritoneal (VP) shunt was placed for hydrocephalus. He developed fever after a few days. The above procedures were done at another institution. The patient came to us after one-and-a-half months with fever and pneumonia. The pneumonia was treated with intravenous Amoxycillin and Clavulnic acid combination. But the fever persisted in spite of clearing the consolidation in the lungs as evidenced by the X-ray reports. On exploration, there was an abscess in the neck, which was drained. Pus was sent for culture and sensitivity yielded no growth. But Ziehl Neelsen’s stain for pus samples showed acid fast bacilli.

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References