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Role of nimodipine in severe diffuse head injury

Sir,

I read with interest the article published by Pillai.S et al,[1] 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,[2] 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,[2,3] 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,[4] where in, its ability to inhibit vasospasm has a significant beneficial effect in reducing neural damage. In this study by Pillai S et al,[1] 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.[5] 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 Clavulinic 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 and VP shunt was removed. The patient developed Cerebrospinal fluid (CSF) rhinorrhea 3 days following shunt removal for which bi-frontal craniotomy and fascia lata duraplasty was done. After 17 days of shunt removal, the abdominal wound was found to be tender, indurated and hence it was debrided. This was followed by left-sided VP shunt insertion another 10 days later.

Provisional diagnosis was pneumonia and infected VP shunt.

Investigations done showed: Hemoglobin-12.2 gm%, CBC-9820 (Neutrophils-76%, lymphocytes-24%). Cerebrospinal fluid (CSF) showed proteins-56 gm% and White blood cells (WBC) count of 20 (30% polymorphs, 65% lymphocytes).

Pus sent for culture and sensitivity yielded no growth. But Ziehl Neelsen's stain for pus samples showed acid fast bacilli

following which, culture on Lowenstein Jensen's medium yielded a growth of *M. fortuitum* in a period of 7 days and anti-tuberculosis drug susceptibility testing showed sensitivity to Kanamycin and Ciprofloxacin but resistance to standard drugs namely Isoniazid, Rifampicin, Streptomycin, Ethambutol, Pyrazinamide, Ofloxacin, Amikacin, Sparfloxacin. The patient was treated with 1 gram of intramuscular Kanamycin once a day for two months and 200ml of intravenous Ciprofloxacin twice a day for three weeks followed by oral 500 mg twice daily for six months.

On discharge, the patient was afebrile, conscious, obeying, moving all four limbs. After six months, there was resolution of lesions and no systemic symptoms.

Mycobacterium fortuitum is an environmental, rapidly growing organism that is found in soil, dust and water. It can colonize without causing invasive disease. It has been implicated in soft tissue infections, osteomyelitis and postoperative infections and injection abscesses.[1] *M. fortuitum* infections have been reported in various surgical procedures.[2-4]

M. fortuitum very rarely causes Central Nervous System (CNS) infection. VP shunt infection by *M. fortuitum* was unheard of till Midani et al[5] reported it in a 13-year-old Spina bifida patient. CNS infection by *M. fortuitum* occurs due to trauma, contamination during surgery or communication with an infected focus. Contamination during surgery is what we speculate for the VP shunt infection in our patient. Amikacin seems to have been successfully used for the treatment of *M. fortuitum* infection. But in vitro drug susceptibility using Lowenstein Jensen's medium by resistance ratio method in our case showed resistance to Amikacin and standard anti-tuberculosis drugs. Kanamycin and Ciprofloxacin were the drugs found to be effective in vitro, that were used for the treatment in our patient after the removal of the shunt and after surgical debridement which is the mainstay of treatment in skin and soft tissue infections.

Thus the possibility of contamination with *M. fortuitum* must be kept in mind while placing ventriculo-peritoneal shunt. So utmost care with regards to aseptic precautions is necessary.

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Intravenous valproate in post-anoxic myoclonic status epilepticus: A report of ten patients

Sir,

Post-anoxic myoclonic status epilepticus (MSE) is difficult to treat. Valproate (VP) is an established antiepileptic drug (AED) with broad-spectrum efficacy in generalized and partial seizures.[1,2] Clinical trials with intravenous VP have shown encouraging results in myoclonic epilepsy and status epilepticus.[3,4] Intravenous VP has been shown to be effective in terminating MSE in a patient with juvenile myoclonic epilepsy.[5] We present our observations on the effect of intravenous VP in post-anoxic MSE.

We studied the efficacy of intravenous VP in 10 patients who developed MSE following anoxic cerebral injury in the peri- and postoperative (within 24-48 hrs) period. The clinical details, the primary disease for which the operation was done, and the type of anesthesia were recorded in all the patients. Informed consent was taken from the relatives for the drug administration. Initial loading dose of VP was 20 mg/kg at a rate of 20 mg/minute. This was followed by 10mg/kg bolus every 6 hours for 24 hours (the total dose in the first 24 hours: 60 mg/kg). Patients were then given a maintenance dose of 10 mg/kg every 6 hours till the patient was shifted to oral VP.[6,7] The infusion was continued till MSE was terminated. The frequency of seizures and the time taken for the termination of MSE were noted. Metabolic (hepatic, renal and pancreatic function tests), hemodynamic (heart rate, blood pressure, respiratory rate and electrocardiographic changes) and hematological parameters were monitored. Arterial blood gases and oxygen saturation were also monitored. Serum valproate levels were not measured.

The clinical characteristics of the patients are given in Table 1. There was a significant reduction in the frequency of seizures in all the 10 patients. MSE was terminated with intravenous VP alone in 6 patients and the time duration for the termination of MSE ranged between 2-10 hours. Four patients needed 30 mg/kg and 2 patients needed 40 mg/kg of VP to terminate MSE. An additional infusion of a second AED, intravenous diazepam or lorazepam, one each was required in 2 patients to terminate MSE. The time taken for the termination of MSE