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 al5 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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References


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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.2,3 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

434 CMYK
was 26 and 38 hours. The remaining 2 patients continued to have seizures, but with a reduced frequency (approx 25% reduction). During intravenous VP therapy, no adverse effects, including reaction at the infusion site, respiratory depression, cardiac arrhythmia or hypotension were observed. There was no change in hemodynamic parameters. Two patients died. Six patients recovered completely and 2 patients were left with significant neurological deficits.

Introduction of the parenteral form has facilitated the use of VP in situations like seizures in the operative or postoperative phase. Several advantages are evident with intravenous VP: a physiological pH, ready-to-use preparation, non-requirement of any organic solvent, no incompatibility with other intravenous solutions and high stability at room temperature. A high success rate of intravenous VP has been observed in various studies.

Excellent control of myoclonus in a patient after cardio-pulmonary arrest, with intravenous VP has also been documented when other antiepileptics had failed. Similar results have been described in post-anoxic myoclonus. It is well tolerated and safe in children with refractory epilepsy and in the elderly, even in the setting of concurrent hypotension, labile cardiovascular function or severe illness. Though the Food and Drug Authority recommends an infusion rate of 20 mg/min of intravenous VP, excellent tolerability has been reported with higher rates.

Anoxic brain injury and MSE are life-threatening and notoriously refractory to AEDs. Their long duration is associated with poor outcome and high mortality, hence must be treated promptly. The available conventional intravenous AEDs may induce or exacerbate hypotension and cause respiratory depression requiring intensive monitoring. Hypotension associated with intravenous AED administration may adversely affect the general cardiovascular status by limiting the timely and adequate delivery of the AEDs (since these complications have to be managed by slowing the infusion rate). The mechanism postulated for the efficacy of intravenous VP is rapid penetration of the drug into the brain tissue. Recently, levetiracetam has been found to be effective in myoclonic epilepsy, which may emerge as an alternative therapy.

Our observations with intravenous VP in post-anoxic MSE are preliminary and the study has many limitations. Continuous EEG monitoring was not done and serum valproate levels were not measured. Larger, multicentric and controlled studies are required for intravenous VP to be considered as first-line therapy.

<table>
<thead>
<tr>
<th>No.</th>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>Operation and Anesthesia</th>
<th>Seizures</th>
<th>Comments</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>60/M</td>
<td>renal Cell Carcinoma</td>
<td>nephrectomy (GA)</td>
<td>MSE partial</td>
<td>hypo tension with diazepam</td>
<td>complete recovery</td>
</tr>
<tr>
<td>2.</td>
<td>27/F</td>
<td>enteric Perforation</td>
<td>iliostomy (GA)</td>
<td>MSE GTCS</td>
<td>brainstem reflexes absent. required ventilator</td>
<td>remains in PVS</td>
</tr>
<tr>
<td>3.</td>
<td>45/F</td>
<td>choleystitis with chololithiasis</td>
<td>laparoscopic cholecystectomy (GA)</td>
<td>MSE GTCS</td>
<td>brainstem reflexes absent.</td>
<td>expired later due to pneumonia</td>
</tr>
<tr>
<td>4.</td>
<td>45/M</td>
<td>chronic renal failure</td>
<td>renal transplant (GA)</td>
<td>MSE GTCS</td>
<td>allograft rejection, hypo tension with diazepam</td>
<td>expired Due to septicemia</td>
</tr>
<tr>
<td>5.</td>
<td>18/F</td>
<td>pregnancy</td>
<td>termination of pregnancy (GA)</td>
<td>MSE</td>
<td>Infusion started immediately</td>
<td>complete recovery</td>
</tr>
<tr>
<td>6.</td>
<td>22/F</td>
<td>pregnancy</td>
<td>termination of pregnancy (GA)</td>
<td>MSE</td>
<td>Infusion started immediately</td>
<td>complete recovery</td>
</tr>
<tr>
<td>7.</td>
<td>48/F</td>
<td>myoma of uterus</td>
<td>Epidural anesthesia termination (SA)</td>
<td>MSE GTCS</td>
<td>brainstem reflexes absent. required ventilator</td>
<td>complete recovery</td>
</tr>
<tr>
<td>8.</td>
<td>32/F</td>
<td>ectopic. pregnancy</td>
<td>termination (SA)</td>
<td>MSE</td>
<td>brainstem reflexes absent.</td>
<td>remains in PVS</td>
</tr>
<tr>
<td>9.</td>
<td>52/F</td>
<td>fibroid uterus</td>
<td>hysterectomy (GA)</td>
<td>MSE</td>
<td>infusion started immediately</td>
<td>complete recovery</td>
</tr>
<tr>
<td>10.</td>
<td>44/M</td>
<td>choleystitis with chololithiasis</td>
<td>laparoscopic cholecystectomy (GA)</td>
<td>MSE</td>
<td>infusion started immediately</td>
<td>complete recovery</td>
</tr>
</tbody>
</table>

MSE—myoclonic status epilepticus, PVS—persistent vegetative state, GA—general anesthesia, SA—spinal anesthesia, GTCS—generalized tonic clonic seizures
Sir,

A 24-year-old man presented on 12/05/97 with history of progressive right facial palsy and conductive hearing loss on the right side. His cranial CT scan revealed a homogenously enhancing isodense mass lesion arising from the middle cranial fossa. Surgery could not be done due to financial problems of the family.

On 03/07/2000, he presented again with the additional problem of ataxia. Repeat CT scan showed a significant increase in the size of the tumor with destruction of the underlying petrous bone (Figure 1). He underwent right temporal craniotomy and excision of the tumor. The tumor was approached extradurally and dissected circumferentially and was excised completely. It was arising from the geniculate ganglion of the facial nerve. The tumor-adjoining cranial nerves were saved. The bony defect in the petrous bone was repaired with temporalis fascia. At three years follow-up, the patient showed moderate improvement in facial function.

The facial nerve is the most frequently paralyzed motor nerve, with 95% of infranuclear palsies due to a pathological process within the temporal bone. Neoplasms account for 5% of all facial palsies and neurinomas comprise only a small fraction of these. Facial nerve schwannomas are postulated to arise from the nervus intermedius and its connection in the geniculate ganglion. As the geniculate ganglion is anatomically located towards the anterior surface of the pyramid, the schwannomas originating here are partially located in the petrous bone and their bulk is in the middle cranial fossa. Approximately 30 cases of facial nerve schwannomas presenting as middle cranial fossa lesions have been reported in the literature. The clinical features depend upon the site of origin of the tumor on the facial nerve and the direction of its growth. The principal clinical features of facial nerve tumors are progressive facial nerve paresis and hearing loss.

The management strategy for facial neurinoma consists of tumor removal and facial nerve reconstruction. The surgical approach to facial neurinoma is selected according to the location and extension of the tumor and state of hearing. In these lesions, the facial nerve should be first identified in the fallopian canal, and the nerve can be followed through the tumor while performing decompression and excision. This technique shall probably enhance the chances of facial nerve preservation or reanimation. The greatest determinant of the outcome of facial nerve reconstruction is the duration and sever-

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Middle cranial fossa schwannoma of the facial nerve

Figure 1: Cranial CT scan axial and coronal cuts showing a homogenously enhancing isodense mass lesion arising from the middle cranial fossa with destruction of the underlying petrous bone.