Examination showed anemia, pregnancy of 26 weeks, and normal cranial nerves, power of 1/5 in the upper limbs, and 1/5 in proximal and 3/5 in distal muscles of lower limbs. There was generalized hypotonia and areflexia with bilateral flexor plantar reflex. There was no sensory impairment or bladder and bowel involvement. During hospitalization she developed bilateral facial weakness, dysphasia and dysarthria with worsening of the limb power to 0/5 on the 15th day. Her respiratory effort weakened requiring intubation and ventilatory support on the 34th day due to inability to maintain oxygen saturation. Tracheostomy was done after the 51st day of illness. Obstetricians were consulted throughout her hospital stay. She delivered a healthy baby of 2.4 kg at 37 weeks of gestation and needed outlet forceps with right mediolateral episiotomy to reduce the duration of the second stage of labor. The baby had two rounds of cord around the neck but cried immediately. The baby was healthy and had no signs of birth asphyxia. The postnatal period was uneventful. Following delivery, the patient showed steady improvement and was weaned off from the ventilator on the 20th postnatal day (94th day of illness). She required intragastric tube feeding up to the 98th day of illness. The patient was treated with plasmapheresis (PP), within 24 hours of admission, consisting of five cycles of PP on alternate days with removal of 750 ml plasma and replacement of 300 ml of 5% albumin solution each time. She was discharged from the hospital after 14.5 weeks when she was partly but significantly dependent for activity of daily life. Her power was 3/5 in the upper limbs and 2/5 in both the lower limbs with generalized areflexia.

The results of the electrophysiological study done on the day of admission, are shown in Table 1. The common peroneal nerve had evidence of conduction block as evidenced by a significant reduction in the amplitude of compound muscle action potential. F waves could not recorded in any nerves. Sensory nerve conduction study was within normal limits.

AIDP is a disorder of presumed autoimmune pathogenesis. It is known to develop at all ages and is reported in pregnancy as well. Reports of treatment of AIDP in pregnancy with IVIG and PP are available in the literature. Our case was unique in the way that we treated this case with PP in the second trimester of pregnancy. IVIG and PP are the treatment of choice for AIDP. IVIG is preferred due to lesser complications as compared to PP. Plasmapheresis has been done in pregnancy for both neurological and non-neurological disorders. Only a single report of AIDP in pregnancy being treated with PP is available in the literature.1 The case we are reporting had AIDP in the second trimester. It is well known that AIDP may worsen in the third trimester of pregnancy just as this patient also worsened. She was treated with PP but she continued to worsen and required ventilatory support. Maintaining vitals, adequate ventilatory support without any complications and termination of pregnancy (by delivery) helped her to recover faster.

V. Goyal,* B. K. Misra, S. Singh, K. Prasad, M. Behari
Department of Neurology, All India Institute of Medical Sciences, New Delhi - 110029, India. E-mail: drvinyagoyal@hotmail.com

Table 1: Electrophysiological study

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Distal Latency (msec)</th>
<th>Amplitude (uV)</th>
<th>Conduction Velocity (m/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Nerve</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rt. Median</td>
<td>4.9</td>
<td>0.77</td>
<td>35.4</td>
</tr>
<tr>
<td>Rt. Median-F wave</td>
<td>Not recordable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rt. CP</td>
<td>5.16</td>
<td>2.49</td>
<td>45</td>
</tr>
<tr>
<td>Rt. CP-F wave</td>
<td>Not recordable</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Death following Ventricular cerebrospinal fluid shunting in supratentorial malignant tumor associated with hydrocephalus

Sir

A 26-year-old male presented (in 1996) with complaints of headaches and right focal motor seizures for two years. The patient was dull and drowsy but had no higher function disturbance or focal neurological deficit. There was bilateral florid papilledema. Computerized tomography (CT) scan showed a large, anterior corpus callosal tumor and moderate hydrocephalus (Figure 1). A biventriculoperitoneal shunt was performed. The cerebrospinal fluid pressure was high and it flowed out like a ‘jet’ through the cannula used during the shunt. Following surgery, the patient could not be

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reversed from anesthesia. Neurological examination three hours after surgery revealed that the patient was deeply unconscious and the left pupil was dilated and fixed. Within an hour, the right pupil also dilated and was unresponsive to light. Cerebral decongestants proved to be of no benefit. CT scan showed that both the ventricular catheters were in-situ and the ventricles were collapsed (Figure 2). The cerebral hemispheres showed extensive edema. The patient succumbed on the third postoperative day. Post-mortem examination showed swollen cerebral hemispheres and generalized cerebral edema.

Discussion

Supratentorial intra-axial tumors lead to a rise in the intracranial pressure by virtue of the tumor mass and its surrounding edema. Occasionally, there is a ventricular dilatation as a result of direct or indirect compression of the cerebrospinal fluid pathways\(^1\). In large hemispheric tumors, which result in a significant rise in the intracranial pressure, contralateral ventricular enlargement is frequently seen. Ventricular dilatation and cerebral edema are often considered to be a part of the pathological process. In our case an unconventional treatment strategy was adopted. A biventricular shunt was done. The aim of such a procedure was to attempt to reduce the intracranial pressure, relieve the patient of his symptoms, to normalize the altered cerebral blood flow and to ‘relax’ the brain that would assist the surgeon in the definitive surgical procedure planned through an interhemispheric approach at a later date. The strategy was unsuccessful and the patient worsened dramatically after the shunt.

We attempted to analyze the cause of the fatal outcome in an otherwise well preserved patient and reviewed the relevant literature. As the shunt surgery in itself was uneventful and the postoperative scan did not show any evidence of hemorrhage, direct trauma by the shunt assembly appeared unlikely. It is known that ventricular tapping and shunt insertion in the presence of a large posterior fossa tumor may result in upward or reverse herniation or may result in subtle movements in the tumor due to a change in the naturally adjusted pressure dynamics following drainage of cerebrospinal fluid from lateral ventricles.\(^3\)\(^-\)\(^5\)

Such movements may be the cause of hemorrhage in some tumors.\(^4\)\(^-\)\(^6\) Spinal tumor movement and impaction in the spinal canal and neurological worsening following a lateral ventricular shunt surgery has been recorded.\(^7\) Posterior fossa, suprasellar and thalamic tumor patients have been recorded to worsen neurologically following a shunt operation.\(^5\)\(^-\)\(^8\) Tumor movement and alteration of the delicately balanced intracranial pressure have been implicated as the cause of the clinical worsening.\(^5\)\(^-\)\(^9\)

Our patient had a large tumor mass. The intraventricular pressure was high as was observed during the ventricular tapping whilst performing the shunt surgery. Both these factors contributed to a significantly high intracranial pressure. The neurological worsening following the ventricular tapping in our case suggests that the rise in the intraventricular pressure was in some manner maintaining or balancing the intracranial dynamics. Following the ventricular cannulation and rapid drainage of the cerebrospinal fluid, there could have been movements within the tumor or of the tumor as a whole. The large tumor could have probably impacted on to the hypothalamus or the internal capsule, or could have resulted in a subtle but definitive midline shift that initiated a chain of events that resulted in increased cerebral edema and later transtentorial herniation. There was no obvious hypoxic event during the induction of the anesthesia. Despite the fact that the cause of worsening in our case could not be actually confirmed, it appears that any form of ventricular drainage prior to surgery on large cerebral tumors
associated with hydrocephalus is detrimental and should be avoided. The role of ventricular dilatation and the impact of a rise in the intraventricular cerebrospinal fluid pressure in the presence of a large intracranial tumor need to be evaluated further.

D. P. Muzumdar, M. G. Bhatjiwale, A. Goel
Department of Neurosurgery, King Edward VII Memorial Hospital and Seth G. S. Medical College, Parel, Mumbai, India.
E-mail: dmuzumdar@hotmail.com

References


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Isolated foot weakness caused by a parasagittal metastatic parotid adenocarcinoma

Sir,

Although the most common cause of foot drop is a lesion of the fifth lumbar root, based upon the homuncular topography of the primary motor cortex, one would predict that a parasagittal cortical lesion might also cause isolated unilateral foot weakness. Surprisingly, a review of the neurological literature reveals a dearth of case reports documenting the existence of this entity. Additionally, the case reports describing primary parotid tumors metastasizing to the brain are few and far between. We describe here the case of a parasagittal metastatic parotid tumor that resulted in isolated foot weakness.

A 60-year-old man presented with left foot weakness of a month's duration. The weakness was of gradual onset, limited to one foot, and was unaccompanied by pain or sensory loss. He had no history of trauma or other neurological symptoms (including sphincter disturbance) and no constitutional symptoms. He had had a right-sided facial mass for seven years for which he denied seeking formal medical treatment.

Physical examination revealed an immobile, non-tender subcutaneous right facial mass measuring 6.5 x 4.5 cm, overlying the parotid gland. The neurological exam revealed a right-sided lower motor neuron facial palsy. He had a left steppe gait and complete paralysis of the ankle and toes of the left foot: all flexors, extensors, evertors and invertors had 0/5 strength. There was no tremor, ataxia, or fasciculations. There was mild hypertonicity of the left quadriceps, with no evident atrophy. He had muscle stretch reflexes that were significant for two beats of ankle clonus on the left, with otherwise diffuse and symmetric 3+ hyperreflexia in all four limbs. The jaw jerk was absent, and there was no abnormal grasp response or other primitive reflex. Plantar reflexes were flexor bilaterally. Sensation was preserved throughout.

Aspiration biopsy of the subcutaneous facial mass was performed, and pathology revealed the lesion to be a poorly differentiated adenocarcinoma of the parotid gland. MRI of the brain with gadolinium revealed a right parasagittal ring-enhancing lesion, with mild surrounding edema. Since the lesion in the brain was single and presumably metastatic, it was resected. Pathology revealed it to be a poorly differentiated adenocarcinoma, with cytological features resembling those of the parotid specimen.

Two aspects of this case were unusual: first, the clinical presentation of isolated foot weakness from a parasagittal cortical lesion, and second, brain metastasis from a primary parotid tumor. The most common tumor type to cause distal foot weakness is parasagittal meningioma, with other reported lesions being astrocytomas, demyelinating plaques, and cranial trauma, which however, the foot weakness in such cases is typically part of a constellation of signs that include some degree of hemiparesis and sensory changes as well as constitutional symptoms such as nausea and vomiting. Suspicion of upper motor neuron disease in such cases is aroused by the presence of a positive Babinski sign labeled “spastic foot drop” by some. Symptoms or signs pointing to an intracerebral etiology, when present, are headache and papilledema. Although adenoid carcinoma metastasizing to the brain has been reported, brain metastases from parotid tumors are rare. There are two case reports of multiple brain metastases from adenoid cystic parotid carcinomas, but these differed substantially from our case in cytopathology, pattern of metastasis (single versus multiple) and clinical sequelae. It has been hypothesized that in the rare cases that parotid tumors do metastasize intracranially, they do so by way of peri-neural or endoneural spread.