Sir,

An anterior sacral meningocele is a cerebrospinal fluid-filled unilocular or multilocular extension of the dura mater and arachnoid mater out of the spinal canal through a defect either in the anterior sacral wall or antero-laterally through an enlarged vertebral foramen or coalesced foramina into the pelvic retroperitoneal and infraperitoneal space. Since its description by Bryant in 1837, only about 154 cases have been reported in the world literature. There have also been reports of an anterior sacral meningocele being a part of the Currarino triad, which is a hereditary condition diagnosed when three abnormalities are noted: an anorectal malformation, an anterior sacral defect, and a presacral mass.

An 18-year-old unmarried female patient presented with a history of gradually increasing difficulty in defecation for two years. On digital rectal examination, a mass was felt in the presacral region. No cutaneous stigma or any vertebral column abnormality was visible. Plain skiagram revealed the characteristic 'scimitar' sign, characteristic of anterior sacral meningocele. Magnetic Resonance Imaging lumbar myelogram confirmed the presence of meningocele. As the meningocele sac appeared to be too large for direct ligation, a thecoperitoneal shunt was performed. Following surgery, the presacral mass could no longer be palpable by digital examination. Subsequent myelograms at 6 months and 1-year intervals revealed gradual reduction in the size of the meningocele sac, although there was no complete obliteration.

Adson advocated a posterior transsacral approach for treating anterior sacral meningocele. Such an approach entails sacral laminectomy and intradural exploration to expose the anterior communication with the meningocele, aspiration of the sacral laminectomy and intradural exploration to expose the anterior sacral meningocele. Such an approach entails.

References

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Blinking of the eye on voluntary movement on the side of paralysis in a case of cerebrovascular accident

Sir

Cerebellar ataxia and hemiparesis are common with lesions in the region of the internal capsule. The palmo-ocular reflex is not so well recognized or reported.

A 40-year-old, non-diabetic or hypertensive male patient was admitted after about 24 hours of sudden onset of slurring of speech disturbance and weakness of the right side of the body. The weakness was more pronounced in the leg. The neurological examination revealed that the power was Grade 4 in the right-sided limbs. The deep tendon reflexes were brisk on the right side and plantar reflex was extensor on the right side. There was no sensory deficit. The finger nose and knee heel test demonstrated ataxia on the right side which appeared out of proportion to the weakness. The patient was observed to have blinking of the right eye when he was asked to move his right hand for passive physiotherapy. The blinking and tremors of the right hand were more pronounced when the hand movements were carried out nearer to the face.

CT scan brain showed a fresh non-hemorrhagic infarct in the territory of the left middle cerebral artery and affecting the region of the ipsilateral corpus striatum and corona radiata. In addition there was an old infarct in the right middle cerebral artery territory affecting the regions of the lentiform nucleus and corona radiata. Another old lacunar infarct was observed in the left lentiform nucleus.

The exact explanation of the cause of the “palmo-ocular”
reflex is unclear. However, affection of frontopontine fibers in the anterior limb of the internal capsule and activation of the red nucleus as an alternative pathway for transmitting cortical signals to the spinal cord (corticorubrospinal pathway) could be the cause. This could also explain the cause of tremors in the right hand which increased in frequency as the hand approached the face, as the red nucleus has a similar relation with the cerebellum as that of the cerebral cortex with the cerebellum.

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Lower end of ventriculoperitoneal shunt embedding in liver parenchyma

Sir,

Insertion of ventriculoperitoneal shunt is one of the commonest neurosurgical procedures. Though a safe and simple procedure, it is not devoid of complications. The common complications associated with shunt surgery are blockage, infection, over-drainage and malfunction.

A 5-year-old female child had a non-communicating hydrocephalus. A Medtronic moderate pressure ventriculoperitoneal shunt was inserted. After about 15 days of surgery, the patient developed headache, vomiting and low-grade fever and mild pain in abdomen. There was referral of pain to right shoulder. X-ray of the upper abdomen showed that the shunt tube was coiled in the right subdiaphragmatic region. Ultrasound abdomen revealed a cystic cavity in the right lobe of the liver with shunt tube inside it. CT scan abdomen (Figures 1) was done, which showed shunt tube embedded in liver parenchyma and a cystic cavity around the tip of the tube. The patient was given preoperative cover of 3rd generation cefalosporin and the lower end was taken out. The shunt tube distal to the chamber was replaced by Chhabra MDR shunt and reinserted through a left inguinal incision. The postoperative period was uneventful. She was asymptomatic at 3 months follow-up and ultrasound abdomen showed resolution of the cyst in the right lobe of the liver.

There are numerous complications of the lower end of the shunt described in the literature. By the above case, the authors want to share their experience of this never before reported complication.

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Klinefelter’s syndrome with myopathy-A case report

Sir,

An 18-year-old male had a decline in the intellectual functions since childhood. The parents also complained of episodic falls and transient loss of consciousness. These episodes occurred on an average, once in every two months, for two years. There was difficulty in rising from the sitting posture.

On examination the patient had marfanoid features. He had small testicles and sparse facial and axillary hair and mild to moderately impaired cognitive functions. Except for bilateral mild flaccidity of calf muscles, there were no other deficits. No obvious behavioral changes were observed.

Routine laboratory investigations showed no abnormality. EEG and cranial CT scan were normal. EMG showed myopathic pattern in all four limbs. Nerve conduction study was