artery (Acom) junction was identified separately from the aneurysm. Excision of the aneurysm along with the vessel was done. Post operatively the patient recovered well with no neurologic deficits. CT head on the following day showed no significant infarctions. Histopathological examination of the aneurysm was suggestive of thrombosed wall of aneurysm with no evidence of inflammation or infection.

OFA aneurysms are rare and have been reported along with associated vascular malformations and multiple aneurysms.[1] We report an isolated case of a partially thrombosed distal OFA aneurysm in a patient with no predisposing factors. As it was partially thrombosed and was arising from a small vessel, preoperatively it was mistakenly thought that it was arising from the A1 segment. However, only at surgery was the exact nature of the aneurysm defined. It is important to distinguish RAH from the OFA. The RAH usually originates within 4 mm of Acom region (most commonly in the proximal 0.5 mm of A2). Usually it is single but rarely can be duplicated or absent.[2] The OFA arises from the A2 about 5 mm from the AcomA junction. It then courses across the gyrus rectus and olfactory tract supplying the orbital gyri, gyrus rectus and the olfactory tract and bulb. Hence, before sacrificing a vessel the above distinguishing points must be kept in mind, it is important to dissect the vessel distally to confirm that it is not RAH prior to sacrifice.

We report a rare case of partially thrombosed OFA aneurysm arising distal to the origin of the vessel in a patient who had no predisposing factors. Such a case has not been described before. Partially thrombosed aneurysms may give a misleading picture of its origin. It is important to distinguish the OFA from RAH prior to sacrifice of the vessel.

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Is staged surgery for giant vestibular schwannomas always better in improving outcome: Needs socioeconomic consideration?

Sir,
Bandlish et al. reported 12 cases of giant vestibular schwannomas (GAS) operated through a retrosigmoid approach in a staged manner over two consecutive days.

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Authors observed that staging improved tumor resection rate and also provided comfort to surgeon without increasing major surgical risks in hospitals; in hospital lacking advanced facility staging cannot be recommended for every case; however, occasional staging is unavoidable and definitely advocated. Optimal management for GVS must balance long-term functional outcome with tumor control. Most surgeons would opine surgical resection as the first-line treatment. Improved understanding of the natural history and pathophysiology of vestibular schwannoma led to possible complete surgical resection. Ramina et al. observed that complete resection of GVS was possible in all primary but also in recurrent and large residual cases in single-stage surgery with a low morbidity rate is possible, but preservation of the facial nerve is difficult due to severe scar tissue. Total surgical removal is the only treatment option for these giant lesions. Samii et al. also further advocated and opined total resection of GVS is possible. Samii et al. evaluated the outcome of radical surgery in giant vestibular schwannoma and concluded total tumor removal can be achieved using a retrosigmoid approach with zero mortality and further hearing preservation is possible. However, it was associated low morbidity rate for facial nerve. GAS management definitely possess a surgical challenge due to giant size of lesion, small capacity of posterior fossa and poor surgical plane with brain stem, small space for surgical manipulation, bleeding and these collectively may contribute to neurological worsening and rare cases morbidity and mortality. With advancement in the field of neuroimaging, delineating extension, anatomical relation of neurovascular structure, intraoperative monitoring, and immense experience of growing number of neurosurgical specialist have yielded insight to deal with challenge in a scientific way. Preparation of surgery starts from a preoperative period, co-morbid illness, presurgical anesthetic evaluation and initiation of perioperative corticosteroid. Proper positioning is very important, as elevated head position gives advantages of sitting position, thereby considerably minimizing blood loss and providing comfort to surgeon ease. Need for CSF diversion should be considered in the form of lumbar drain, VP shunt, EVD drain, or releasing CSF from cistern and putting cottonoid patty into cisterna magna aids in continuous drainage of CSF making posterior fossa lax.

Intraoperative larger craniotomy may be helpful to avoid deleterious effect of prolonged retraction and foramen magnum rim should be exposed and removed to aid in CSF release and dissection of migrated tonsil and lower pole of lesion after defining sigmoid and transverse sinus, and mastoid drilling. Dura is first incised inferomedially to release CSF from a cistern magna, arachnoid is liberally opened, and tonsil is retracted up and medially. Intraoperatively debulking with large biopsy forceps is done without peeling arachnoid; as it is seldom associated with considerable bleeding, which can be controlled by bipolar or topical hemostatic agents, gel foam, surgical tamponade with cottonoid. Important to note is that mobilizing arachnoid without proper debulking causes tear of thin venules and vein, which fails to contract in view of stretched wall encasing large residual making persistent stretching of arachnoid, which promotes blood loss. Another important aspect is dissection plane with brainstem vessel medially, after debulking it becomes easy. So principle should be internal debulking and continuing internal debulking and in the end tumor tissue instead of capsule mobilization should be gradually and progressively peeled away from arachnoid coverings, it eases pressure on vessel, which easily contract and promotes hasty hemostasis and further microneurosurgical dissection technique can be vividly applied. So we feel staging may be beneficial in short term, especially in developing country where resource is important constraint. Surgeon scan further improve surgical result with acquiring current surgical technical advancement instead of 2 days surgery proposed by Bandlish et al. on presumption of non-availability of CUSA and facial nerve monitor. Proper positioning, proper exposure and meticulous microsurgical dissection techniques helped us immensely to reduce blood loss, thereby providing opportunity for better dissection and preservation of neural tissue. We are operating with lateral position with head elevation and dissection technique lead to remarkable reduction in need of blood transfusion. Further intraoperative anaesthesia with total intravenous anesthesia without using nitrous oxide or volatile anesthetic agent aid in reducing intracranial pressure, making surgical procedure smooth and proper maintenance of anesthesia avoid intermittent rise of hypertension or raised intracranial pressure for smooth progression of surgical procedure.

Also, the role of trained surgical team including staff nurse and paramedics is vital. So team approach with modern microsurgical technique can go a long way in replicating the result with zero percent mortality although some morbidity is indispensable. As developing countries have scarcity of medical facility, getting overburdened with huge patient load, carry ethical morale to provide care to needy patients, more so staged procedure will definitely warrant double resources and timing and also depriving other patient to undergo surgical procedure.
Isolated hemimegalencephaly in an adult

Sir,

There are a few reports of hemimegalencephaly (HME) in adults; we are reporting a case of isolated HME in a male adult.

A 30-year-old right-handed male patient presented 3 years back with complaints of recurrent episodes of seizures for the past 26 years. Seizures were right focal motor seizures with secondary generalization with a frequency of 10-15 episodes per month initially. There was a history of gradual onset of weakness in right upper and lower limbs. He also had intellectual disability. On examination, patient did not have any cutaneous markers to suggest of neurocutaneous syndrome. He had dysarthria, mini-mental state examination score was 13/30 and Addenbrooke’s Cognitive Examination (52/100), right side upper motor neuron type facial nerve palsy, and mild right-sided spastic hemiparesis with extensor plantar response.

Electroencephalogram (EEG) was grossly abnormal, there was diffuse background slowing with theta and delta activity with almost continuous bursts of spike/polyspikes, sharp, and slow wave discharges over left hemisphere [Figure 1]. Magnetic resonance imaging (MRI) of brain [Figure 2] showed enlarged left frontoparietal lobe with smooth thickened cortex and pachygyria, indistinct grey/white differentiation, subcortical white matter showed hyperintensity on T2-weighted axial image [Figure 2a], hypointensity on T1-weighted coronal image [Figure 2b], and hyperintensity on Fluid Attenuated Inversion Recovery axial image [Figure 2c], straightening of the left frontal horn. He was treated with oral sodium valproate 10 mg/kg body weight initially, and gradually the dose was increased to 20 mg/kg body weight to control seizures. During the follow-up period of two and half years, there was no recurrence of seizures.

HME is a severe developmental malformation of the brain, remarkable for its extreme asymmetry,[1] Localized megalencephaly accounts for one quarter of all HME cases and predominantly seen on the left side (72.7%).[2] It is divided into three forms: (1) Isolated form, most common (66%); (2) syndromic form associated with several neurocutaneous syndromes; and (3) total HME, less common.[1] The classic neurological triad includes intractable seizures with onset typically within the first few months of life is the most common presenting symptom, contralateral hemiparesis, and severe psychomotor delays.[2,3] Causes of HME may be related to insults as early as the third week of gestation. One of the mechanisms of pathogenesis of HME is a disorder of cellular lineage and establishment of symmetry that occurs around the third week of gestation.[3,4] Recently, the etiology of HME was demonstrated as somatic mutation of AKT3.[4,5] The common EEG pattern is an asymmetrical background activity and sporadic wide spikes and/or spikes-waves complexes are usually confined to the malformed hemisphere. Other two less common patterns are the unilateral suppression burst and unilateral hypsarrhythmia over the abnormal hemisphere.[1,5] MRI of brain reveals the following: Unilateral enlargement of one or part of the cerebral hemispheres; with a thickened cortex; broad gyri, polymicrogyria, or agryria; shallow sulci; indistinct grey/white differentiation; increased volume and T2 signal of white matter;

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