Cerebellar hemorrhage following endoscopic third ventricular colloid cyst decompression

Sir,

Cerebellar hemorrhage following supratentorial surgery is a rare complication with only 29 cases
reported in the literature. However, there has been no reported case of cerebellar hemorrhage following endoscopic surgery.

We present the case of a patient aged 45 years, a known hypertensive and hypothyroid on treatment developed features suggestive of raised intracranial pressure. On examination there were no focal neurological deficits. Computed tomography (CT) was suggestive of a colloid cyst [Figure 1].

He underwent endoscopic fenestration and decompression of cyst. The cyst contents were drained completely but the capsule was partially excised due to the dense adhesions with thalamostriate vein. The surgery was uneventful. Five hours after surgery patient suddenly became drowsy with shallow respiration. He was intubated and underwent an emergency external ventricular drainage. The computed topography (CT) showed a well-organized hematoma measuring $5 \times 2.7 \times 2.5$ cm in the left cerebellar lobe with no mass effect [Figure 2]. The coagulation profile was normal as were the routine biochemical and hematological parameters. The patient’s blood pressure was normal. In view of the normal neurological status and clot volume of 17 ml it was decided to continue with the conservative management.

A repeat CT done on postoperative day 7 showed a persisting hematoma with no change in size. However, in view of persistent headache along with surrounding edema, hydrocephalus along with effacement of basal cisterns and fourth ventricle, a decision to evacuate the hematoma was taken. A left paramedian suboccipital craniectomy and evacuation of hematoma was done along with a ventriculoperitoneal (VP) shunt in the same sitting. Patient was discharged in normal neurological status with no motor sensory deficits.

Patient developing remote infratentorial hematoma after supratentorial surgery is a rare complication with only 29 cases available in the reported literature. We did an extensive literature review including our own experience of 96 cases with endoscopic decompression of colloid cyst but could not find a similar case.

The mechanism of cerebellar hemorrhage after supratentorial surgeries is a matter of debate and is probably multifactorial. Various factors have been proposed which include the following causes.

Intracranial hypotension due to sudden decompression of the ventricles might lead to a cerebellar hematoma, as there could exist a pressure differential between the two compartments. Other predisposing factors suggested include an altered coagulation profile, but most reported cases including ours had a normal coagulation profile and postoperative hypertension. An important proposed factor is overdrainage of cerebrospinal fluid (CSF) intraoperatively or postoperatively via drains. In all the patients, the occurrence of hemorrhage was associated with loss/removal of large amounts of CSF either intraoperatively or postoperatively. Another postulated cause has been excessive rotation with hyperextension of head leading to obstruction of internal jugular veins.

In our patient, the plausible mechanism could be a combination of sudden supratentorial decompression leading to intracranial hypotension with resulting displacement of the cerebellum. Various management strategies have been suggested but the most important indicators for surgery include the neurological status of the patient, the presence of hydrocephalus, the size of the hematoma and the extent of fourth ventricular and brainstem compression. The reported mortality and morbidity has been high (33 and 58%).

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Presumed paraneoplastic brainstem encephalitis secondary to ovarian teratoma

Sir,

Ovarian teratomas are rarely associated with paraneoplastic central nervous system (CNS) disorders, especially in adolescent girls.[1,2] Benign ovarian teratomas have been associated with paraneoplastic CNS disorders in only two cases, one each of paraneoplastic brainstem encephalitis (PNBE) and paraneoplastic encephalomyelitis.[3,4]

We present the second case of PNBE associated with a benign ovarian teratoma and the first in an Indian adolescent.

A 15-year-old girl was evaluated for intractable vomiting of four weeks duration. Examination revealed an abdominal mass and ultrasound of the abdomen suggested a juxta-ovarian mass. She underwent laparoscopic surgery and complete removal of the mass. Histopathology revealed a mature ovarian teratoma with cystic components. Her vomiting persisted one week after surgery. Postoperative abdominal ultrasound and upper gastrointestinal (GI) endoscopy were normal. Two weeks after surgery, she complained of difficulty in walking and slurred speech. On examination, she had dysarthria and a wide-based gait. Routine blood tests were normal. Non-contrast magnetic resonance imaging (MRI) brain and nerve conduction studies were normal. A repeat contrast MRI two weeks later was also normal. Cerebrospinal fluid examination was normal. At this point, paraneoplastic brainstem encephalitis was considered. She was given a course of IV methylprednisolone (IVMP) 1 g/day for five days. Her vomiting abated after three days. Her ataxia and dysarthria improved slowly and she was able to ambulate. Four weeks after treatment, she was able to function normally with minimal residual dysarthria. Her gait ataxia had nearly disappeared. The parents were unwilling for paraneoplastic antibody screening (anti Ro and anti La). At follow up, one and a half years later, she was asymptomatic.

Our case is unique in two respects. It is the first case of presumed paraneoplastic neurological disorder associated with an ovarian teratoma reported from India. Secondly the presenting manifestation of paraneoplasia was intractable vomiting. Only after other cerebellar signs developed was it possible to diagnose a paraneoplastic syndrome.

Before diagnosing a paraneoplastic disorder, it is mandatory to rule out other conditions such as CNS metastases, infections, chemotherapy or radiation-induced symptoms, loco-regional tumor complications, autoimmune conditions and CNS demyelination. An MRI study helps in excluding many of these conditions.

Detection of antibodies that co-localize with EFA6A may be helpful in the diagnosis of these syndromes.[5] However, in a developing economy like ours, these tests are expensive and the cost saving can often be utilized for therapy. This case report emphasizes the necessity of considering a PNBE when neurological symptoms or intractable vomiting develop in the presence of an ovarian teratoma. It should be noted that steroid responsiveness was observed in an earlier case of paraneoplastic encephalomyelitis associated with a benign ovarian teratoma.[4]

Steroid responsivity and prolonged or permanent remission seem to be a feature of PNBE associated with ovarian teratomas. Clinicians should consider steroids when teratoma removal alone does not result in alleviation of symptoms.

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Accepted on 25-09-2007