Comment on dreaded complications of mistaken identity: Hygroma versus effusion following decompressive craniotomy

Sir,

We read with interest the article “Dreaded complications of mistaken identity - hygroma versus effusion following decompressive craniotomy” by Rambarki and Rajesh[1] and commend the authors on highlighting the complication of residual collection causing mass effect on the brain after cranioplasty.

However having encountered such cases ourselves, we disagree with the contention that it was the nature of the fluid within that predisposed to this complication and hold that the encysted collection that was not freely in communication with the cerebrospinal fluid (CSF) that led to this occurrence.

Subdural hygromas are relatively common following decompressive craniotomy (21-50%)[2] and it is arachnoid breach that is the cause of these collections. The fate of these collections is one of the following:

- In the vast majority of patients, the hygroma resolves with time and the scalp flap tends to sink in prior to cranioplasty. The time frame for the process of subdural hygroma formation has been described by Aarabi et al.[3] as peaking in the 3rd to 4th week and disappearing by 14-17 weeks after surgery
- Some patients have small residual collections that disappear following cranioplasty. This is because cranioplasty tends to restore the dicrotic wave form of CSF pulsations that are lost following decompressive craniotomy.[4] Restoration of this waveform aids in the absorption of CSF though arachnoid granulations (opening of which is pressure dependent)
- In a small minority, an in the case illustrated, an encysted collection is formed. These collections expand with ingress of CSF through a one-way valve till the time intracystic pressure reaches a level high enough to prevent further inflow of CSF. The skin is invariably stretched as is seen in the computed tomography (CT) image of this case. Any attempt to tap this fluid percutaneously invariably results in a recollection. Moreover even an intraoperative lumbar puncture as was done in this case, to make the flap lax and allow proper seating of the bone flap merely drains ventricular and cisternal CSF and does not address the root issue of the encysted collection. This is borne out by the fact that if the collection in this case had been in free communication with the CSF space, xanthochromic fluid would have come out through the lumbar puncture too.

The authors noted xanthochromic fluid at second surgery and regretted their inability to have identified this preoperatively as an effusion rather than a hygroma furthermore stating it resulted in the “dreaded complication.” However, the preoperative CT scan was innocuous, and it is obviously not feasible to get a magnetic resonance imaging study done in every case. Moreover, bleeding within subdural hygromas is a well-known entity that has been described previously.[5,6]

In order to prevent this complication, we must have an appreciation of the fact that large, tense and longstanding collections are not “routine hygromas.” In addition, we must appreciate that these collections will recur following cranioplasty irrespective of their content unless certain actions are taken, such as closing the point of ingress of fluid or opening the cyst widely to allow communication with surrounding spaces. The inner layer of the cyst is adherent to the pia and often the opening through which CSF leaks cannot be found. We do not recommend a routine hunt for the site of the leak. If the point of the leak is seen, however, coagulation with bipolar at a low setting or application of fibrin glue and piece of muscle or artificial dura may help.

Alternatively, thick sections of the outer layer (as the authors encountered in their case), may be excised, fluid let out, and the bone flap replaced. This is akin to incising the dura and treating a chronic subdural hematoma. There will be some subgaleal collection in the postoperative period as the fluid comes out through the craniotomy margins, but this invariably gets absorbed over time. We fail to see the benefit of repeating a duraplasty since the outer layer of the collection is not involved in the pathogenesis of this collection.

Subsequent recollections may also be treated by the burr hole aspiration or a subduro-peritoneal shunt rather than going through the process of reopening the bone flap.
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References


Opsoclonus – Myoclonus syndrome induced by phenytoin intoxication

Sir,

Phenytoin is an antiepileptic drug that continues to be used widely for treatment of focal and generalized tonic-clonic seizures. Phenytoin has narrow therapeutic window. Therefore, even a minor increase in dose leads to toxicity. Opsoclonus and myoclonus both are adverse effect of phenytoin [1] but opsoclonus–myoclonus syndrome (OMS) as a consequent effect of phenytoin toxicity is not previously reported in the literature.

Figure 1: Photograph of the oral cavity showing gum hypertrophy

A 19-year-old male presented with complaints of acute onset abnormal, episodic intermittent involuntary jerky movement of all four limbs, trunk and eye lids without loss of consciousness from 12 days. These movements used to subside during sleep and not exacerbated by visual, auditory or tactile stimulus. There was positive history of difficulty in walking with swaying on either side and slurring of speech from last 10 days. On general examination he had gum hypertrophy [Figure 1] but no evidence of skin rash. Neurological examination revealed abnormal chaotic multidirectional movement of eyes suggestive of opsoclonus with evidence of myoclonic jerks involving all four limbs, face and eyelids [Videos 1 and 2]. He had severe trunk as well as gait ataxia, incoordination and dysmetria. Rest of neurological examination was normal.

He had past history of tuberculous meningitis, hydrocephalus, and generalized tonic clonic seizures since the age of two and half years. He was treated with complete course of antitubercular therapy along with ventriculo–peritoneal shunt for hydrocephalus. His phenytoin dose was increased from 300 to 500 mg/d 1 month back due to one episode of GTCS and Tab Levetiracetam 500 mg BD was added recently for myoclonic jerks but there was no improvement in his symptoms. A clinical diagnosis of OMS with ataxia (gait, truncal as well as appendicular) was considered.

Hematological, biochemical and cerebro spinal fluid examinations were normal. CSF was negative for Herpes simplex virus (HSV), Japanese encephalitis, measles and varicella zoster as well as Cryptococcus neoformas, and Mycobacterium tuberculosis. The immunological study for Dengue, rubella, HIV, Mycoplasma pneumoniae, Rickettsia, HCV, HBV, Ebstein–Barre virus, poststreptococcal infection and coxsackie infection...