Commentary

Persistent accumulation of fluid in the subdural space may be either hygroma or hematoma or effusion.[1] Subdural effusion (SDE) following decompressive craniectomy for traumatic brain injury constitutes one of the common late complication.[2] SDE may show spontaneous resolution or enlarge with passage of time. It usually runs a benign course and most commonly resolves spontaneously. SDE's natural course is self-limiting showing spontaneous resolution with passage of time leading to resolution of mass effect or very rarely may have slow progression requiring neurosurgical intervention.[3,4] Only few reported cases needed surgical intervention as reported by authors, however, recognition of such cases is very important to avoid neurological deterioration. Seung *et al*. analysed 89 cases who underwent decompressive craniectomy for traumatic brain injury and reported that a total of 29 cases developed SDE, and all cases were managed conservatively and showed spontaneous resolution except for one case who needed surgical intervention for progressive mass effect and failure to resolve.[4]

Postulations for increase in volume of SDE include rupture of arachnoid membrane with permitting cerebrospinal fluid collection or continuing transudation leading to increase in volume and accompanying rise in the intracranial pressure. Further progression may be accelerated due to rupture of bridging veins causing hemorrhages collecting in to subdural space, with passage of time formation of neo-membrane and recurring micro-hemorrhages can cause further expansion and mass effects, responsible for compression of adjacent neural tissues causing global or focal neurological deficits.[2,4,5]

Various risk factors responsible for rare cases showing failure to resolve and necessitating single or multiple surgical interventions include bilateral presence of subdural hygroma, associated postoperative pneumocephalus, poor neurological status on admission, and heterogeneous consistency. According to Miyake *et al*., SDE can be divided into four distinct clinical stages in sequential phases: Initial stage rupture of arachnoid membrane; followed by intervening stages of synthesis of inner membrane; next being inner as well as outer membrane formation stage; and subdural hygroma or hematoma formation.[6]

Treatment modalities including pediatric cases are tapping of anterior frontanelle, single stage burr-hole, burr-holes with drains placed in subdural space kept for few days, drain placement following simple twist drill, and even simple craniotomy.[1,4-6] Although every therapeutic surgical option has some limitation, frontanelle tapping can be used in infants for all stages of SDE and being a minimal invasive and massive hemorrhage is very rare occurrence provided punctures are carried out using small needle. However, continuous external drainage following burr-hole or twist drill potentially carry a risk of meningitis and usually advocated to be kept for less than 5 days.[6] However, in cases requiring prolonged drainage, an Ommaya reservoir placement and repeated tapping under aseptic precaution or even endoscopic evaluation of SDE cavity and burr-hole irrigation areother useful alternatives. However, Miyake *et al*. discourages use of shunt placement between SDE cavity and peritoneal cavity.[6] Subdural shunts have an inherent long-term potential complication, including malfunction, infection, and some cases get shunt dependent due to development of subarachnoid to subdural cerebrospinal fluid (CSF) communication and, it may need second surgery for its removal, if not required.[5] According to Klimo *et al*., the most suitable method for surgical treatment of SDE should have greatest probability of definitively managing as a single stage surgical intervention having minimal chance of postoperative complications and without need to put a shunt.[5]

Rambarki *et al*. reported an interesting case of SDE, detected at time of replacement of craniotomy flap, lumbar drain was placed in the preoperative period with temporary relief and following surgical procedure for bone flap replacement developed

hemiplegia, computed tomography (CT) scan showed reaccumulation of SDE with evidence of fresh bleed necessitating reexploration craniotomy with decompression of loculated SDE and bone flap removal in order to control raised intracranial pressure.[7] Satyarthee *et al*. also reported a rare interesting case of progressive massive SDE in a 10-month-old male infant, who despite bilateral burr-hole placement and drainage, after discharge from hospital presented with progressive diminution of visual acuity and massive scalp bulge at placed burr-hole sites, mimicking rabbit ear sign, necessitated subduro-peritonreal shunt desperately in a prophylactic attempt to prevent impending scalp swellings rupture.[1] So timely detection of such progressive cases of SDE, although very rare incidence, is crucial and appropriate measure can avoid catastrophic neurological sequlae.

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