A 73-year-old male patient presented in an unconscious state with weakness of the right side of the body, 8 hours after a fall. On examination, he had no eye opening, no verbal response, and on pain was localizing with the left hand. He was flexing weakly with the right upper limb on pain. There was no history of antiplatelet or anticoagulant intake and he had no other preexisting medical illnesses. Computed tomography (CT) scan showed a left-sided acute subdural hematoma (SDH) with significant mass effect, effacement of cisterns, and midline shift (►Fig. 1A–C). There was no contralateral intracranial bleed or injury. He underwent left-sided temporoparietal craniotomy, durotomy, and clot evacuation. Intraoperatively, an arterial bleeding point was noted in the left frontal lobe and was cauterized. The brain was extremely lax and hence after duraoplasty, the bone flap was replaced. Postoperatively, he was reversed from anesthesia but not extubated and was noted to have left-sided focal seizures. A CT scan was done that showed contralateral acute SDH with a small interhemispheric SDH with no parenchymal contusion (►Fig. 2A–E). However, as this was not causing any significant midline shift or cisternal effacement conservative treatment was continued. A magnetic resonance imaging (MRI) study was done later too that showed only an acute subdural hematoma on the right side with no cerebral injury (►Fig. 3A–D). His seizures were managed with anticonvulsants and serial CT scans showed resolution of the contralateral SDH over time (►Fig. 3E–H). He was discharged uneventfully after 3 weeks with a full recovery.

Discussion

Contralateral hematomas after cranial surgeries are rare but potentially life-threatening complications.1,3 The most commonly described type of contralateral hematoma is an extradural hemorrhage.4 Contralateral intracerebral hemorrhages (usually expansion of a preexisting contusion) are the second most common5 and contralateral acute SDH is the rarest. Most of the reports in literature on development of a contralateral acute SDH after operation are in the nature of case reports. However Chrastina et al6 found a contralateral acute SDH, necessitating reoperation in 5 out of 86 cases operated for an acute SDH while Shibahashi et al7 have stated that the incidence and mortality of contralateral acute SDH after ipsilateral acute SDH evacuation to be 4.1 and 75%, respectively.

Sudden shift of the contralateral hemisphere toward the side of the craniotomy after decompression with rupture of bridging veins,2 bleeding from a previously undetected contusion,8 release of tamponade of a contralateral bleeding point,2,9 sudden increase in blood flow in the contralateral side after rapid release of intracranial pressure at the time of decompression,2,8,10 bleeding diathesis secondary to coagulopathy or due to intake of antiplatelet or anticoagulant medications,2 faulty positioning during surgery causing cerebral
venous hypertension (due to excessive neck rotation), and overuse of osmotic dehydrating agents have all been postulated as reasons for development of a fresh contralateral acute SDH.

Most cases described in literature underwent decompressive craniotomy initially for the acute SDH where the potential of contralateral hemispheric shift toward the craniectomy defect is greater, unlike in our case where the bone flap had been replaced. Suspicion of a contralateral pathology was precipitated in our case due to development of focal seizures on the same side as the craniotomy while other cases in literature were unmasked following imaging necessitated by unexplained neurological worsening or inexplicable postoperative deficits and in those, where there is suspicion of contralateral associated intracranial injuries for early detection and treatment of this complication.

Conclusion

It must be remembered that whatever the pathogenesis, contralateral acute SDH is more likely in elderly patients than an extradural hematoma since their dura is intimately adherent to the skull, and further these patients have brain atrophy with a potentially larger subdural space where the bridging veins may rupture as the brain shifts intraoperatively. It is mandatory to have a low threshold for early postoperative imaging in elderly patients, those with unexpected intraoperative brain bulge or inexplicable postoperative deficits and in those, where there is suspicion of contralateral associated intracranial injuries for early detection and treatment of this complication.

Conflict of Interest
None declared.

References