The early and aggressive management of extradural hematomas (EDH) is a classical teaching during neurosurgery residency.1-3 Posttraumatic seizures (PTS) after EDH share a significant percentage of causality, but apparently remain a less studied entity. The major literature analyzing EDH and PTS dates back to the late 20th century and there are only a couple of recent studies.4-7 Additionally, an often-debated issue is the role of prophylactic antiepileptic drugs (AEDs) in patients suspected of having high chances of developing PTS.8,9 Uniform use of AEDs in all cases can lead to drug-related side effects and complications and in some cases unnecessary AED dependence.9,10

An online search PubMed database was performed by using literature and using the search strategy “((“extradural hematoma”[All Fields] OR “hematoma, epidural, cranial”[MeSH Terms] OR (“hematoma”[All Fields] AND “epidural”[All Fields] AND “cranial”[All Fields]) OR “cranial epidural hematoma”[All Fields] OR (“extradural”[All Fields] AND “hematoma”[All Fields]) OR “extradural hematoma”[All Fields] AND (“epilepsy”[MeSH Terms] OR “epilepsy”[All Fields])) AND (“seizures”[MeSH Terms] OR “seizures”[All Fields]) AND (“anticonvulsants”[All Fields] OR “anticonvulsants”[MeSH Terms] OR “anticonvulsants”[All Fields])” on PTS after EDH returned only a handful of articles (►Fig. 1).4,5,7 Three studies were excluded as there was no clear categorization of intracranial hematomas,11 diagnosis of extradural hematoma not clearly mentioned,12 and no clear description of seizure groups.13

One of the early reports was by Bryan Jennett from the Institute of Neurological Sciences, Glasgow in 1975.6 The peculiarity of this report was that the case series was of the pre-CT era and surgical interventions were based only on clinical findings. Among patients of seizures due to posttraumatic intracranial hematomas (excluding chronic subdural hematomas), EDH was reported as a cause of early seizures (within 1 week of head injury) in 10% (15/146) and late seizures in 22% (13/59) patients. Jennett also found out that only 2% of early PTS patients had an evolving EDH. The next significant report came in 1991 by Jamjoom et al from Bristol.4 They categorized EDH patients with epilepsy in two subgroups, based on CT findings into those with exclusive EDH and those with other intradural traumatic insults.4 Although they found the overall incidence of late epilepsy to be 6%, in the pure EDH group, it was only 2% as against 17% of those with additional intradural damage. Another data analysis from a multicentre North American TBI database of 795 patients from 1989 to 2000 was reported by Ritter et al in 2016.7 Among the various findings, EDH was found to be the cause of early and late PTS in 14.5% and 16.9%, respectively. The most recent report on the incidence of PTS due to EDH came from the series of 484 TBI patients by Pormontree et al from Thailand in 2019.6 The authors analyzed early PTS in TBI patients from April 2017 to March 2018. Twenty-seven patients (5.6%) had early PTS due to various intracranial insults. Among these, EDH was found to have an adjusted odds ratio of 3.98 on multivariate analysis (p value = 0.001).

PTS is a known complication of head injury.6,7,10,14-17 Whether they occur in the early (within a week) or in late posttraumatic period, this sequela of brain injury can significantly deteriorate the quality of life and is considered an independent factor.5,7,14-17 All the contemporary studies on
posttraumatic epilepsy (PTE) are in patients with intradural injuries.\textsuperscript{10,16,18} Hence, considering the significant share of PTE attributed to EDH in the tune of 15 to 20%, there is an emergent need to undertake a well-formulated study to understand the exact correlation in the current advanced imaging era and then accordingly tailor the prophylactic antiepileptic treatment.

None declared.

References