Perihematomal edema

Besides the initial hematoma volume, secondary hematoma expansion, intraventricular hemorrhage and mechanisms of secondary brain injury—e.g. the development of a perihematomal edema (PHE) –are responsible for the high morbidity and mortality. PHE, being part of the secondary brain damage in ICH, develops partly due to thrombin-mediated endothelial cell damage and inflammation and red blood cell lysis. Therapies like hypothermia or continuous infusion of hypertonic saline aim to reduce PHE. Aggressive lowering of the blood pressure also decreases absolute PHE growth, at least in patients with volumes of intracerebral hemorrhages of about 10cc.

Decompressive craniectomy is associated with a significant increase in perihematomal edema compared to patients who have been treated conservatively. Perihematomal edema itself lasts about 60 days if it is not treated, but decompressive craniectomy ameliorates the mass effect exerted by the intracerebral hemorrhage plus the perihematomal edema, as reflected by the reduced midline shift [1)].

1)
Fung C, Murek M, Klinger-Gratz PP, Fiechter M, Z'Graggen WJ, Gautschi OP, El-Koussy M, Gralla J, Schaller K, Zbinden M, Arnold M, Fischer U, Mattle HP, Raabe A, Beck J. Effect of Decompressive Craniectomy on Perihematomal Edema in Patients with Intracerebral Hemorrhage. PLoS One. 2016 Feb 12;11(2):e0149169. doi: 10.1371/journal.pone.0149169. eCollection 2016. PubMed PMID: 26872068; PubMed Central PMCID: PMC4752325.