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Intraparenchymal Hemorrhage, Treatment Strategies and Surgical Outcomes

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Introduction

Spontaneous İntracranial Hemorrhage (ICH), non- traumatic hemorrhage into the brain parenchyma ± ventricles, is a severe type of stroke with high mortality rates (Figure 1) [1]. Systemic arterial hypertension and cerebral amyloid angiopathy represent the two main risk factors of primary ICH [2,3]. Although ICH is a destructive disease for the cerebral tissue, there is no specific treatment that has been shown to improve outcome. Main target of the terapies is about hematoma expansion including decreasing blood pressure, the management of tranexamic acid and using of recombinant activated factor VII [4,5]. However all of theese have failed to improve functional outcome [6,7].

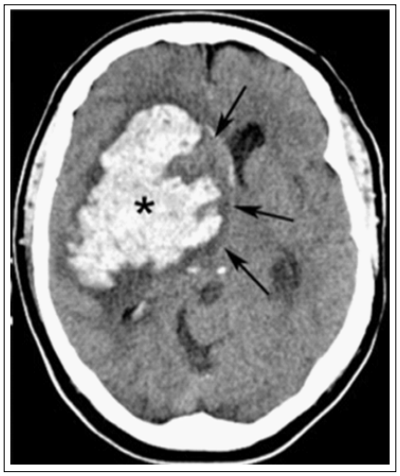

Figure 1: Intraparenchymal Hemorrhage with Midline Shift

Patients may have different symptoms depending hematoma volume and location. For instance, patients who have a temporal lobe located hematoma may suffer seizure, whereas patients with hematoma located in frontal lobe may be asymptomatic. Larger hematomas lead to life-threatening circumstances by causing herniation syndrome.

Hematoma evacuation may have therapeutic potential but its blood products into the surrounding healthy brain parenchyma. The most common sites of spontaneous ICH are the deep brain structures, including the basal ganglia and the thalamus, a large layer of brain tissue must be crossed during surgery, which may cause iatrogenic damage of healthy cerebral tissue.

Surgical options for ICH patients

- Hematoma removal via open craniotomy (most common approach),
- Decompressive craniectomy ± hematoma drainage,
- Stereotactic endoscopic aspiration,
- Minimally invasive catheter evacuation followed by thrombolysis.

Mechanisms of Brain Injury after İntracerebral Hemorrhage

There are 2 types of mechanisms of brain damage includes primary and sedepending ICH includes the primary effects of blood into the brain parenchyma and the secondary effects of hemoglobin breakdown and its products. Initially, there is the direct effect of acute hemorrhage into the brain parenchyma, causing disruption and mass effect within the cerebral tissue. This primary brain injury is followed by the interruption of bleeding in approximately two thirds of patients. How- ever, in the remaining one third of patients, hematoma continues to expand in the first 24 hour, which contributes to additional mass effect, midline shift , leading to further neurological deterioration and an increased risk of unfavorable outcome [8, 9,10].

Hematoma volume and location are the two main predictors of outcome related to the hematoma itself [9-11]. Hematomas larger than 30 ml are statistically associated with unfavorable outcome [12,13]. The patients with hematoma volume larger than 60 ml and aGCS lower than 8 are predicted 30-day mortality higher than 90%. Acute hematomas larger than 150 mL generally leads to death by the reason of the sudden increase in intracranial pressure and consequently the reduction in cerebral perfusion pressure below critical levels [13].

Additional to the physical effects of the initial and expanding hemorrhage, there are the effects of persistent hematoma and its blood products leading to a complex cascade of events (Figure 2) [14-16]. The majority of ICH patients may not require surgery; however, the common idea isearly surgical removal of an intraparenchymal hematoma could be beneficial for the patients. This benefit is based on the assumption that clot removal would recover the cerebral tissue restoration, reducing mass effect and correcting midline shift, and therefore it would improve cerebral perfusion

by decreasing intracranial pressure. Additionally, hematoma drainage could prevent or at least cut into the cascade of secondary brain injury (Figure 2) owing to the deleterious effects of hemoglobin and its products into the brain. However, the surgical removal of a blood clot within the brain is not riskless. In order to reach the hematoma deep brain structures and a large layer of healthy cerebral tissue usually needs to be dissected, it is generally performed under general anesthesia. Additionally, postsurgical complications, such as hemorrhages, infarctions and infections, are not uncommon in this clinical scenario, which increase the rates of outcome with mortality morbidity [17,18].

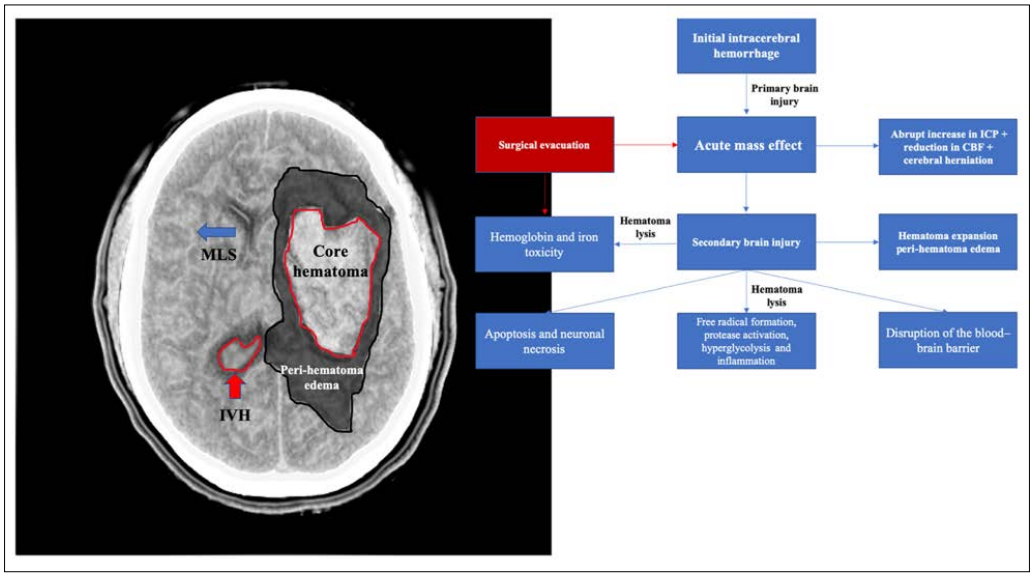

Figure 2: Mechanisms of Secondary Brain İnjury after ICH. MLS - Midline Shift; IVH - İntraventricular Hemorrhage

Besides open craniectomy with hematoma removal, decompressive craniectomy with or without hematoma evacuation may be an other aproaching for patients in coma (Glasgow Coma Scale Score < 8) with significant midline shift and large hematomas, or patients with refractory intracranial pressure.

Dikici and Colluoglu showed in their study that hematoma volume and midline shift of the patients who underwent surgery due to ICH improved significantly (Table 1). They also evaluated ICH patients who tested positive (9 patients) and negative (73 patients) for Covid-19 disease in two groups. Though there were no significant differences mortality rates between two groups, both mean and median survival time was longer for the patients without Covid-19 [19].

Table 1 : The Paired Analysis of Preoperative and Postoperative IPH Results

Variables	Preoperative	Postoperative	p-value
Glasgow Coma Scale Score	7.5 ± 3.5	7.5 ± 4	0.926
Hematoma volume (cm^3)	82.4 ± 42.5	23.7 ± 44.8	0.0001
Midline shift effect (mm)	9.3 ± 4.7	5.2 ± 5.1	0.0001

Conclusion

Open craniotomy for early hematoma drainage after intracranial hemorrhage is the common approach. There is biological plausibility based on the prevention of cerebral herniation, the control of intracranial hypertension, and also avoidance or at least reduction in the impact of blood and its products on surrounding healthy tissue. Craniotomy for hematoma drainage remains a life-saving measure in critical situations. Larger than 30 ml hematoma volume may cause unfavorable outcome. If hematoma volume is larger than 150 ml or larger than 60 ml with lower than 8 GCS score, mortality may reach at the level of 90 %. Decompressive craniectomy without hematoma evacuation can be performed fort he patients in coma (GCS score < 8).

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