Bedside Intracranial Hematoma Evacuation and Intraparenchymal Drain Placement for Spontaneous Intracranial Hematoma Larger than 30 cc in Volume: Institutional Experience and Patient Outcomes

Tyler Carson D.O., Vladamir Cortez D.O., Dan E. Miulli D.O.

Department of Neurosurgery, Arrowhead Regional Medical Center, Colton, CA
Email: tyler.a.carson@gmail.com

Nontraumatic intracranial hemorrhages (ICH) occur for a number of reasons including uncontrolled hypertension, amyloid angiopathy, anticoagulant use, cerebrovascular disease, tumors, migraines and those that occur after invasive procedures. Bleeding is usually short-lived and is tamponaded by anatomical and physiological means however is associated with a 30 day morbidity and mortality of 60% and 30% respectively.[1] Elevated blood pressure defined as a systolic pressure greater then 140 mmHg is seen in 75% of patients with acute ICH and strict control of blood pressure is paramount in prevention of delayed rebleeding. [2] The ICH score described by Hemphil in 2001 gives us a good predictive factor for the predicts 30 day mortality.[3]

Many attempts to classify surgical indications for evacuation of ICH have been and continue to be studied. The international surgical trial in intracerebral hematoma (STICH) looked at the outcome of 1033 patients from 83 centers in 27 countries patients treated with early surgery (open craniotomy) vs. initial conservative treatment.[4] Ultimately, the STICH trial showed no significant difference between early surgical treatment and non-surgical treatment groups as a whole. However, there was a subset of the early surgical group which seemed to have a better outcome than conservatively treated patients. These patients had supratentoral ICH that came within 1cm of the surface. To further investigate these findings the STICH II trail was performed

which looked at early surgery (within 48 hours) vs. initial conservative treatment specifically for patients with ICH with volume of 10-100 ml, within 1 cm of surface, without IVH and a GCS motor score of 5-6 and GCS eye opening score of ≥ 2.[5] The results showed there was no increase in death or disability at 6 months between groups and there was a small benefit in overall survival.

Clearly there is a subset of patients that benefit from evacuation of hematoma. To that end the MISTIE and MISTIE-II trials (Minimally Invasive Surgery Plus Recombinant Tissue type Plasminogen Activator for Intracerebral Hemorrhage) looked to employ a minimally invasive approach to clot evacuation in hopes to decrease the clot size and perihematomal edema (PHE) more effectively than medical treatment alone.[5,6] Currently the MISTIE III trial is underway and will determine if the reduction in PHE and clot size results in improved neurologic outcome. Here at ARMC we have employed similar techniques, but adapted them to be performed bedside in an ICU setting rather than under general anesthesia in the operative room.

Objective:

The Goal of this study is to discuss the institutional experience and patient outcomes over the past 3 years in placement of bedside intraparenchymal drains for intracranial hematomas larger than 30 cc in size.

Methods:

Beginning in October 2014 our institution began placement of intraparenchymal drains for patients with intracranial hematomas greater than 30 cc in volume and not related to

aneurysmal or AVM rupture. With this study a retrospective review of patient data was performed to determine the effectiveness of IPH drain placement. The primary outcome measure was improvement in GCS from presentation to post treatment. Secondary outcome measures were reduction in clot size, actual versus predicted mortality, re-bleeding associated with catheter placement or after rtPA administration and catheter misplacement requiring repositioning.

The technical placement of the IPH drain utilizes bony anatomical landmarks referenced from CT head to localize the entry point for trajectory of drain placement. The entry point is also chosen keeping in mind where the clot comes closest to the cortical surface, preferably 2-3cm from the Sylvain fissure, midline, or venous sinuses and avoiding eloquent brain tissue such as motor strip. Hair is clipped and sterile preparation of surgical site performed. After local anesthetic and appropriate conscience sedation is administered a 3 cm incision carried down to the cranium. Using the hand twist drill 2 holes are created in same incision ~ 2 cm apart one hole directed toward center of IPH. The dura is opened at both holes. A brain needle with stylet is inserted into clot keeping in mind trajectory and depth at which clot will be encountered based on CT imaging. Once clot has been accessed, the tract is slowly dilated by rotating the needle in a progressively wider circular motion. A Frazier suction tip with stylet is inserted along the tract then the stylet is removed. The wall suction is then connected to the Frazier tip and turned on to 90-120 mmHg suction. The clot is then aspirated until approximately 50% of clot remains based on the output from wall suction. The suction is then turned off and Frazier sucker removed. A trauma style ventricular catheter is then passed down the tract into center of hematoma and tunnel > 5cm from insertion site. The drain is secured to skin and incision is

closed. The catheter is connected to blub reservoir but left off suction initially. A post procedure CT Head is obtained to ensure catheter is in the center of the hematoma cavity. If no active bleeding is noted and catheter is in an acceptable position then 2 mg rtPA are administered through the catheter immediately upon return to ICU and the drain is clamped for 1 hour then opened to bulb suction. The CT head is repeated after 12 hours and if no increase in size of hematoma we proceed with administration of 2 mg rtPA per catheter every 12 hours clamping for 1 hour after each administration. It is important to maintain strict SBP goal of <130 mmHg during rtPA administration . The administration of rtPA is continued until output from the catheter is minimal or CT head showing clot size < 15 cc volume.

Results:

A total of 12 patients were treated from October 2014 to December 2016, see Table 1. Informed consent was obtained from family member. All patients were treated in the ICU at Arrowhead Regional Medical Center via the method described above. All procedures were performed by a neurosurgery resident. Of the 12 patients, 6 patients had the procedure performed by a single surgeon. The remaining 6 were performed by various surgeons. One patient had care withdrawn per family request after the drain was placed and was withdrawn from the study.

The average number of days treated with the drain was a mean 6.4 days and median 5 days. The number of days treated varied based on the resolution of the hematoma.

The patient's level of consciousness was tracked throughout the hospital course via Glasscow Coma Scale score (GCS). The median initial GCS on arrival was 8T. The lowest GCS that received

treatment was a patient who presented as a GCS 5T and the highest was a GCS 12. The median GCS after treatment was 11T. The GCS on arrival and post treatment was compared using the student t-test and P value was calculated to be 0.094 which did not reach statistical significance.

The average clot size on admission was 70.87cc with a range of 26.25 cc to 113 cc. After treatment the average clot size was reduced to 15.95cc, a reduction of 76.9% on average. See figures 1 & 2 which show presenting CT head, CT head post drain placement and CT head post treatment course. The student t-test was performed and p-vale was calculated to be 0.0000035 which was statistically significant.

Additionally, all patients showed decreased 30-day mortality when compared to predicted 30 day mortality based on ICH score on arrival. There was one incidence of rebleeding which stabilized and treatment was continued. There were 2 incidences of drains requiring repositioning.

Conclusion:

The benefit of evacuation of ICH hematomas remains a controversial subject. Some argue that evacuation is only indicated in life threatening situations such as impending herniation due to the morbidity and mortality associated with craniotomy without evidence of significant neurologic improvement. Other studies indicated that a reduction in mass effect from evacuation of ICH results in decreased perihematomal edema (PHE) and decreased secondary injury similar to the viable penumbra in the case of ischemic stroke. Ideally the answer to both cases is a minimally invasive surgical approach that reduces morbidity and mortality yet results

in reduction of PHE. The MISTIE trail attempts to address this with minimally invasive surgical clot evacuation. Our study takes this concept a step further by performing minimally invasive clot evacuation in an ICU setting at the patient's bedside. By performing this surgery bedside we have been able to limit our patient's exposure to general anesthesia, which in many cases results in patient remaining intubated post-op and the sedative effects of general anesthetics.

The results for our series of 12 patients shows a trend towards improvement in GCS after treatment with minimally invasive intraparenchymal clot evacuation and drain placement at bedside, though it did not reach statistical significance. There was a reduction in clot size after treatment, which was statistically significant. In addition, a single case of re-bleeding was noted and 2 cases of catheter placement that required repositioning. These, however did not affect the reduction in clot size post treatment nor result in a drop in mental status. In addition, the 30-day mortality actually observed in our patients was lower than that estimated using ICH score

Based on our experience this procedure can be safely performed at the bedside. The use of AXEM or other frameless stereotaxy may be beneficial in the novice surgeon performing this procedure to avoid misplacement of the catheter. This procedure was extremely effective in reducing clot size, and data trends indicated there may be benefit to improvement in neurologic status. Ultimately more patients need to be treated with a longer follow-up period to determine the effectiveness of this technique.

References:

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Age	M/F	Etiology	Days tx	ICH sc	GCS inital	GCS post	Clot Adm	Clot post tx	Chng Clot%	Mort D30
49	M	BG Hemorrhage	10	3	8T	13	81.2	15.2	81%	No
57	M	BG Hemorrhage	5	3	8T	10T	56.79	8.68	85%	No
77	M	BG Hemorrhage	8	2	9T	11T	95	12.64	87%	No
53	M	BG Hemorrhage	4	3	8T	11T	72.6	12.2	83%	No
69	F	BG Hemorrhage	4	2	12	14	59.3	15.6	74%	No
18	F	Venous infarct	5	N/A	5T	6T	54.4	9.2	83%	No
73	M	BG Hemorrhage	5	2	10	11	113	23.74	79%	No
58	M	BG Hemorrhage	10	3	6T	10T	80	14.85	81%	No
20	F	Venous infarct	4	N/A	8	14	26.25	9.66	63%	No
43	M	BG Hemorrhage	9	3	6T	11T	54	25	54%	No

Table 1

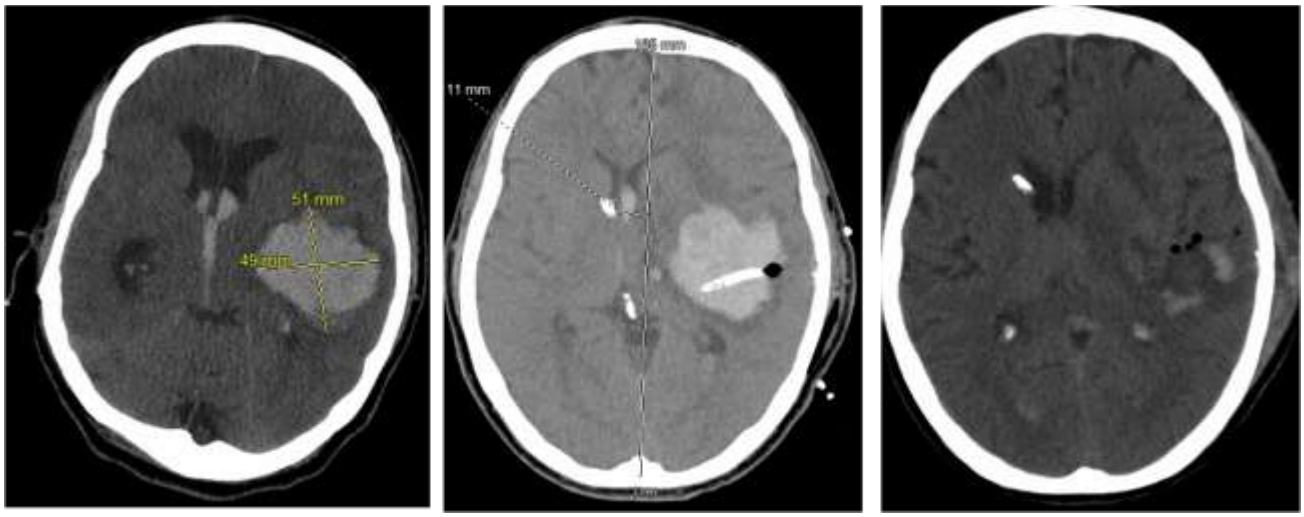

Figure 1

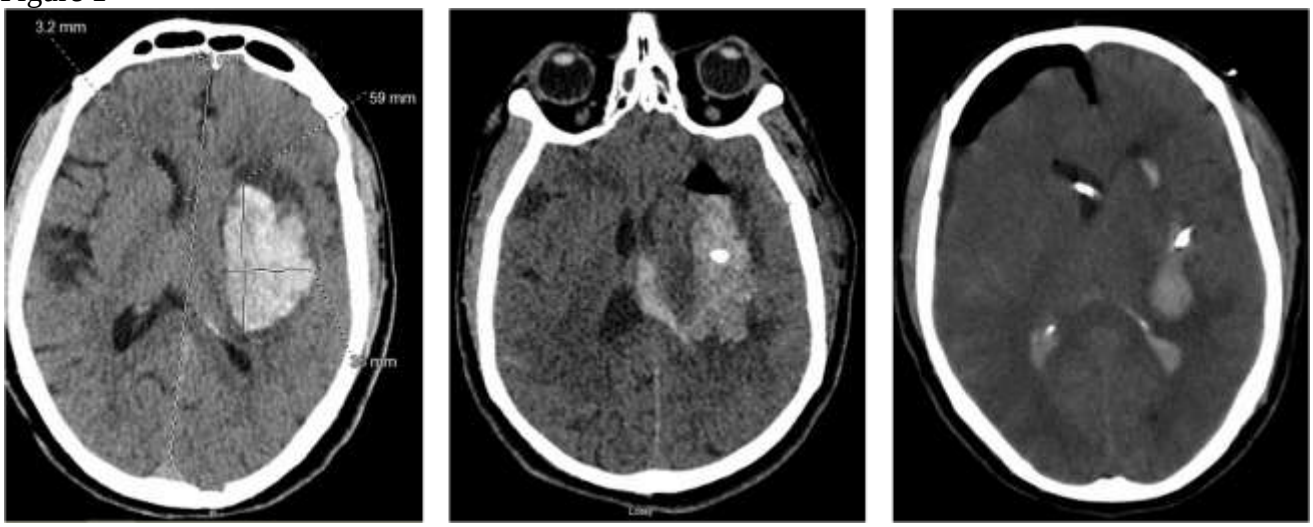

Figure 2