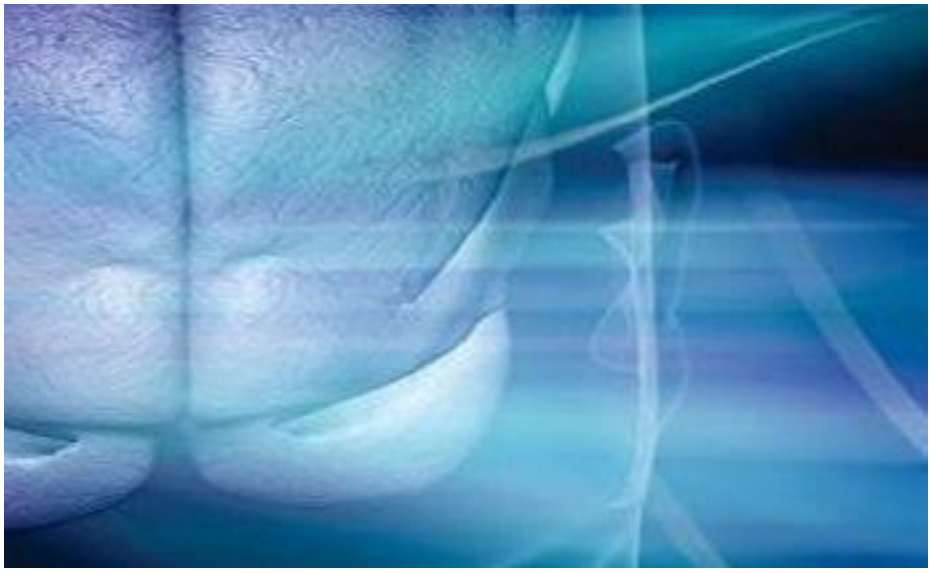

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INSTRUCTIONS FOR AUTHORS

Papers submitted should be original documentation, including photographs. The papers should be single column, double-spaced. The title should be in title case and bold, followed by Authors, degree, organization and city, state.

The papers should contain an abstract and be separated into sections with bold typing of the section title. The page set-up should be 0-6.5 inches. Paragraphs should be indented 0.5 inches. All tables should be submitted separate from the paper. If possible make the tables up to 3 inches wide so that they could fit into a column. This will allow quicker scanning and preparation.

References should be numbered, tab, name of authors. Title of paper. Journal. Year volume:pages.

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EDITOR'S PAGE

Physicians in training, learn and practice research "To formulate, ingrain, and measure, a method of thought, investigation, and evaluation necessary for physicians to have multi-lateral information exchange and communication with experts in areas of scientific and medical discovery, knowledge, and analysis, in order to continuously and efficiently improve human health and patient care." Understanding and performing quality research provides students and residents the tools to propel quality medical care into the community and into the future.

Welcome to the Journal of the American Organization of Neurological Surgeons and the American College of Osteopathic Surgeons Neurosurgical Section. This volume is composed of the Residents' annual papers that were submitted but not published elsewhere. It is therefore dedicated to the future Neurosurgeons and their education. All papers were reviewed by the peer review committee and selected for awards. The papers submitted are excellent, representing some of our talented colleagues. Issues will be published annually. I hope that this issue will spread the knowledge of our residents and our section. We will continue to solicit annual papers and all papers submitted at the annual meeting. This is your Journal paid for by your annual dues. This issue is available on our website AOANeurosurgery.org. This is your organization; please support it as you can.

Thank you,

Dan Miulli, D.O, F.A.C.O.S
Editor

2011 Annual Resident Achievement awards

1st place Douglas Stofko, D.O. for his submission "Retrospective Study of Heparin administration for ischemic stroke when there is an IV-TPA contraindication A Safety Analysis" from PCOM

2rd place Leslie Lyness, D.O. for her submission "Intracranial Hemorrhage on Repeat Head CT" also from PCOM

3rd place Alexandra Beier, D.O. for her submission "Rotary Subluxation: Experience from The Hospital for Sick Children" from Providence St. John's

The papers will be presented during the ACA Meeting Sept 14, 2011 Hilton Atlanta, GA

Retrospective Study of Heparin administration for ischemic stroke when there is an IV-TPA contraindication

A Safety Analysis

Douglas Stofko, DO, Erol Veznedaroglu, MD, FACS; Kenneth Liebman, MD, FAGS
Stroke and Cerebrovascular Center of New Jersey, Capital Health System, Trenton, New Jersey

Abstract

Background: Most patients who are diagnosed with an ischemic stroke arrive after the 3 hour time frame allowed for IV TPA administration. Currently, there are not any well documented and researched immediate treatment options to begin as these patients are being transferred or in a waiting phase for images or surgery. This study is looking to fill the void of administering intravenous weight based heparin as a treatment option for patients who present within 24 hours post ischemic stroke but with a contraindication to IV TPA,
Methods: The study was designed to test whether an anticoagulation regimen of intravenous heparin, in patients with a contraindication to IV TPA, administered within 24 hours of an acute ischemic stroke could be effective and safe. A retrospective review of 257 patients, 223 which received intravenous heparin within 24 hours post ischemic stroke. Pretreatment National Institutes of Health Stroke Scale (NIHSS) and detailed medical history were analyzed. Heparin was started with no bolus and infused at a rate to maintain activated partial thromboplastin time ration 2.0 to 2.5 x control for average of 4 days. The primary endpoint was to analyze patient outcome. This will be done by analyzing length of stay, medical complications during hospital stay, location of discharge compared to pre-stroke residence and NIHSS pre and post admission. Safety end points are bleeding complications, which will be examined until patient improves or is discharged
Results: A total of 257 stroke patients were included. 223 (86.8%) received only IV heparin. Days on IV heparin ranged 1-18 days with an average of 4 days. Length of stay was 1-36 days with an average of 8 days. 52 patients received endovascular procedures (23.3%), 2 patients received subsequent endovascular or cranial procedures (0.9%), 2 patients received only cranial procedures (0.9%) and 167 patients received no surgery (74.9%).
Conclusion: As can be deduced from a survey of the literature, there is a necessity for published data regarding immediate treatments for patients presenting with an IV WA contraindication. Administration of intravenous heparin within 24 hours of an ischemic stroke seems to be safe and warrants further investigation.

Key Words: cerebral ischemia I anticoagulation 11 stroke I heparin

Introduction

While heparin is widely used for treatment of acute stroke, currently there are few clinical trials that have proven efficacy of intravenous heparin in the treatment of stroke (2, 7). Long term studies have been done to show that intravenous heparin administration within 3 hours of acute nonlacunar hemispheric cerebral infarction has shown benefit based on long term outcomes (2). However, the remaining data is sparse and tends to allow long intervals from the onset of stroke to the initiation of heparin or focus on heparin dosing following IV TPA (5, 8, 10, and 14). Nonetheless, heparin has been shown to prevent thrombus propagation which is a potential reason for progressive stroke (1, 4). Currently, there are not any well documented and researched immediate treatment options available to patients not eligible to receive IV TPA. As can be seen from the survey of literature, there is a necessity for published data regarding immediate treatments for patients presenting with an IV TPA contraindication.

Methods

We retrospectively (January 2009 - April 2011) analyzed consecutive patients diagnosed with acute ischemic stroke who did not receive IV **TPA** but received intravenous heparin within 24 hours of, ischemiic stroke onset.

Out of 257 consecutive, stroke patients at our stroke center, 223 patients met the inclusion criteria. The primary, end points of the study were to analyze patient outcome and safety. Patient outcome was evaluated by analyzing length of stay, medical complications during hospital stay, location of discharge compared to pre- stroke residence and pre- and post admission NI HSS. Safety was evaluated by early bleeding complication rates.

Inclusion criteria consisted of patients 18-90 years old with the diagnosis of acute ischemic stroke, symptoms of stroke onset could be narrowed to a specific time and included patients who had a contraindication to IV TPA. Exclusion criteria were any patients who received IV TPA or diagnosed with a hemorrhagic stroke.

Patients matching inclusion criteria were administered weight based intravenous heparin, with no initial bolus, within 24 hours of stroke symptoms, Partial thromboplastin time (PTT) ratio was obtained 6 hours after infusion was started and then everyday to maintain a PTT goal of 2.0 to 2.5 times the control. Heparin infusion was immediately discontinued in case of any hemorrhage. After admission cat scan (CT) of the head, further CT's where only checked on an as needed basis when cerebral bleeding was suspected. Extracranial bleeding was recorded and intravenous heparin discontinued when clinically indicated.

Results
Of 257 consecutive acute ischemic stroke patients, from January 2009 to April 2011 at one single stroke center, 223 patients (86.8 %) met the inclusion criteria. Included were 100 women and 123 men, with a mean age of 66 years (range, 20 — 91).

A total of 257 stroke patients were identified of which 223 received only IV heparin. Days on IV heparin ranged 1-18 days with an average of 4 days. The average PTT was 73 (range, 25-91). 52 patients received endovascular procedures (23.3%), 2 patients received subsequent endovascular or cranial procedures (0.9%), 2 patients received only cranial procedures (0.9%) and 167 patients received no surgery (74.9%).

Received Endovascular &	**2**	0.90%
Received only **Cranial**	2	0.90%
Received Endovascular	52	2.ngici
Received NO Surgery	167	74.89%

Patient outcome was analyzed by evaluating length of stay, location of discharge and N1HSS pre and post ischemic stroke. Length of stay was 1-36 days (with one outlier staying 77 days) with an average of 8 days. Table 1 shows location of discharge compared to pre-stroke residence. 107 patients (48%) where discharged to rehab from home pre-stroke, 42 patients (18.8%) admitted from home were subsequently discharged to home, while another 22 patients (9.9%) were discharged to home with home care. 12 patients (5.4%) died.

Hemorrhagic complications are seen in table 2. Hemorrhagic complications of the brain were differentiated into asymptomatic or symptomatic according to the guidelines of the NINDS trial (12). Symptomatic hemorrhage was seen in 2 patients, while 8 patients had asymptomatic hemorrhage.

Extracranial hemorrhage occurred in the gastrointestinal tract (5 patients), groin site of femoral sheath (2 patients), hematuria (1 patient), rectal bleeding (1 patient), rectus sheath

hematoma (1 patient) and carotid hematoma (1 patient). No deaths resulted from extracranial bleeding.

Table 1

Pre Stroke Location	Discharge Location	Total	%
Assisted	Rehab	[redacted]	0.45
Home	Home	42	18,83
Home	Home Care	22	9.87
Home	Hospice	7	3.14
Home	Hospital	6	2.69
Home	Passed	[redacted]	5.38
Home	Rehab	[redacted]	47.98
Home	Unknown	1	0.45
Homc Care	Rehab	1	0.45
Hospital	Hospital	3	i 35
Rctiab	Kebab	5	[redacted]
Rehab	Horne Care	1	0.45
Rehab	Hospice		0.90
Hospital	Rehab	1	0.45
Long term	Rehab	2	0.90
Nursing	Hospice	2	0.90
Nursing	Nursing Horne	1	0.45
Nursin$_g$	Rehab	[redacted]	0.90
Unknown	Hospice	1	0.45
[redacted]	Rehab	4	1.79

Hemorrhagic Complications of the Brain: Table 2

	N (%)
Symptomatic	*2* (0.3%6)
Fatal	2 (C .9%)
Non Fatal	0
Asymptomatic	8 (8.0%)

Discussion

Our study suggests administering intravenous weight based heparin as a treatment option for patients who present within 24 hours post ischemic stroke but with a contraindication to IV TPA is both safe and warrants further investigation. Currently, the evidence on administering intravenous heparin to patients with acute ischemic stroke is scant. Unfortunately, randomized controlled trials have not studied heparin in patients who have no other treatment options.

The utility of heparins in ischemic stroke is based on the principal that it prevents thrombus propagation. Thrombus formation, propagation or embolization causes new neurological worsening or deficits in 20 — 30% of ischemic strokes (3). Also, heparin has proven to have ant-inflammatory properties, which is known to take place after ischemic stroke (6). With the anti-inflammatory effects of heparin and prevention of thrombus propagation, the use of IV heparin would seem to be a rationale treatment for acute stroke. However, few studies have shown a net clinical benefit to intravenous heparin administration. The majority of the published data does not emphasize heparin administration within 24 hours. The International Stroke Trial (1ST), the largest heparin trial, heparin was administered subcutaneously and not intravenously, therefore PTTs were not closely monitored (10). Long term, randomized controlled trials have been done to show that intravenous heparin administration within the first 3 hours of acute non lucunar hemispheric cerebral infarction has helped patient outcomes based on the 90 day modified Rankin Scale (mRS). However, patients in this time frame would have been available for IV TPA therefore not addressing the patient population that would not be eligible for this option. In the TOAST trial, a low molecular-weight hepannoid was given by intravenous infusion within 24 hours of acute stroke symptoms for 7 days resulting in reduced recurrent strokes and very favorable outcomes were significantly higher in patients given hepranoid vs placebo (14).

Intravenous heparin as well as IV TPA administration has been associated with an increased risk of symptomatic intracranial hemorrhages. However our rate at 0.90% for symptomatic intracranial hemorrhages was significantly lower than previous reported for both intravenous heparin and IV TPA thrombolysis. Other trials of thrombolysis have reported higher intracranial hemorrhage rates but the thrombolysis was given up to 6 hours from onset of symptoms. No other information is available for intravenous heparin clinical trials (5,8).

While there have been contributions to the evidence base by studies such as 1ST, and TOAST and even fewer contributions of heparin administration within 24 hours,

there is still no large published trial of monitored, unfractionated heparin administered within 24 hours in acute ischemic stroke. Our published data supports that a future randomized controlled trial would be safe and is needed to validate the use of intravenous heparin in patients with contraindication to IV TPA.

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Incidence of Enlarging intracranial Hemorrhage on Repeat Head CT
Leslie Lyness, DO, PCOM

Abstract

Objective: To review the literature on traumatic Intraparenchymal Hemorrhage (IPH), to evaluate the incidence of increase size of intracranial Hemorrhage (ICH) that appears on the second CT scan, and to predict the risk of an increase in the hemorrhage size by type of hemorrhage.

Method: In a retrospective chart review of 73 patients at a single Level 1 Trauma Center, the size of the ICH (subdurals, epidurals, and contusions), age, sex, and time differential between the initial CT scan and the second CT scan was evaluated.

Results: A total of 73 patient images from 2008 and 2010 were reviewed, of those, 39 meet the criteria to *be* included in the review. 3 patients were evaluated more than once due to multiple types of hemorrhages, and therefore, the total number of hemorrhages evaluated was 43. 12/43 (27.9%) bleeds were considered to be larger in size with a significant change in the size of the hemorrhage, and of these, 4/12 (33.3%) images had measurements that were larger on the 2" CT, but the radiologist read the scan as "stable". 31/43 (72.1%) images showed no significant change on the second CT, and of these, 14 (45.2%) hemorrhages were actually smaller. 19/43(44.2%) bleeds were contusions; 10/19 (52.6%) of the contusions increased in size on the second Cr. 3/43 (7.0%) bleeds were [OH; 1/3 (33.3%) increased in size. 21/43(48.8%) bleeds were SOH; 1/22 (4.5%) increased in size.

Conclusion: The incidence of increase in size of ICH is greatest for intraparenchymal contusions (52% of contusions increase in size) and least for subdural hematomas (4.5%). in a literature review of Traumatic Brain Injury and IPH, despite the incidence of increase in size of hemorrhages, if a patient has a normal neurological exam, the increase in size of the ICH, regardless of type, rarely, if ever leads to neurosurgical intervention.

INTRODUCTION

The incidence of closed head injury is approximately 200 per 100,000 which amounts to nearly half a million patients annually. Each year, approximately 400,000 pts with head injury are admitted to the hospital for evaluation[2], and the financial burden in the United States for Traumatic Brain Injury (TI31) is estimated to be 100 billion dollars annually[3]. The study of choice for evaluation of any patient with a traumatic brain injury is a CT scan of the head; this study is a relatively quick and easy way to evaluate for the presence of intracranial hemorrhage and skull fractures. There has been some debate over the last few years as to the need for repeat serial CT scans in TM. This study reviews the literature on repeat Head CT in relation to traumatic intracranial hemorrhage, and retrospectively reviews ICH at a Level 1 Trauma center in order to determine the incidence of enlarging ICH on repeat CT.

PATIENTS AND METHODS

At a single Level 1 Trauma center, a retrospective review of 73 head trauma patient images from 2008 and 2010 were analyzed for type of hemorrhage, age, sex, and time differential between the initial and repeat CT scan. The intracranial hemorrhages studied included Subdural Hematomas (SDH), Epidural Hematomas (EDH), and Intraparenchymal Contusions. Subarachnoid hemorrhages and tentorial subdural hematomas were excluded from the study due to the fact that with these hemorrhages it is sometimes difficult to quantify the initial size and amount of enlargement of the hemorrhage. Of the 73 patients, 39 met the criteria to be included in the review. Exclusion criteria were subarachnoid hemorrhage, tentorial suladurals, if only one CT was performed, and if the pt subsequently went to surgery based on the initial CT scan. Three patients had more than one type of ICH (2 patients had 2 types of hemorrhages — SDH as well as a contusion, and one patient had a SDH along with both frontal and temporal contusions), therefore, of the 39 patients, 43 hemorrhages were evaluated. For each image, the measurement of the hemorrhage was performed by the author. If there was > 1.5mm difference in the ***size*** of the

hemorrhage, then the hemorrhage was considered to be increased in size. The images were checked with the radiologists reading and in 4 cases the hemorrhages were measured as larger, but the radiologist's reading was "stable", In one case, the hemorrhage was read "larger, but the measurement was not increased in size.

RESULTS

CT scans of 39 patients were reviewed and of these 39 patients, 43 ICH's were evaluated owing to 3 patients with multiple types of hemorrhages. 31/43 (72.1%) images showed no significant difference in the size of the hemorrhage, and of these 14/31 (45.2%) were actually smaller on the second CT scan. 12/43 (27.9%) of the hemorrhages were considered to be larger, with > 1.5mm increase in the size of the bleed, however, 4 of these hemorrhages (33.3%) were read by the radiologist as "stabler' on repeat CT. In breaking down the images in types of hemorrhages, 19 (44.2%) were contusions, 3 (7%) were epidural hematomas, and 21 (48.8%) were subdural hematomas. Ten out of 29 contusions (52.6%) increased in size on the second CT, 1/3 (33.3%) of the epidural hematomas increased on the second CT, and 1/22 (4,5%) subdural hematomas increased on the second Cr (Appendix 1). The majority of patients were male (71.8%) and there was approximately an even distribution of patients 65 years of age or older versus less than 65. Seventeen patients greater than or equal to 65 years old and 22 less than 65 years old (See Table 1). For each type of hemorrhage, there were more men than women, and hemorrhages affected patients less than 65 years old except for the subdural hemorrhages in which the age grouping was about the same (See Table 2).

The average time between image one and two was 8.7 hours with the range being from 3 hours to 22 hours_ The Median number was 7 hours. In looking at the hemorrhages that increased in size, the range was 3 —18 hours with the average being 7.3 hours. There were two images in this group of 11 scans that were outliers in terms of time between scans (18 hours and 14 hours) while the

average of the other 9 scans in this group was 5.4 hours. These two scans were of patients where the only injury was a contusion, This shorter average time between scans may indicate that due to injury or the appearance of the injury on the initial CT scan, the Neurosurgical staff believed these injuries were somehow more concerning in nature and an earlier repeat scan was indicated.

TABLE 1

	Total (39 patients)	**Percentage**
Male	28	**71.8%**
Female	**11**	**28.2%**
Greater or equal to 65 yr old	**17**	**43.6%**
Less than 65 yr old	**22**	**56.4%**

TABLE 2

	Contusion (# of patients)	**Epidural Hematoma (ft of patients)**	**Subdural Hematoma (It of patients)**
Male	**14**	**2**	**16**
Female	**5**	**1**	5
Greater or equal to 65 yr		1	11
old			10
less than 65 yr old	**13**		

DISCUSSION

In the course of following patients with Traumatic Brain Injury, it is difficult to predict what will worsen over time either clinically or radiographically just from observing the intracranial injury diagnosed on the initial CT scan. There have been articles published concerning this topic, with conclusions made about prognostic factors and who should have repeat CT scans. Although in some cases, the results prove to be

conclusive, the majority of Trauma centers that follow the limited established literature are small which is likely due to the retrospective nature of most of the studies. In terms of prognostic factors influencing the likelihood of progressive hemorrhagic injury, Oertel looked at 142 patients with a mean GCS of 8 of which 60 had progressive hemorrhagic injury. 51% of intraparenchymal hemorrhage, 22% of EDH, 17% of subarachnoid hemorrhages (SAH), and 11% of SDH progressed. In analyzing the prognostic factors that would lead to progressive injury, male sex, older age (> 50 years old), increased PTT (partial thromboplastin time), shorter time from injury to initial CT scan, and the absence of hypotension post-injury correlated positively[4]. Chang also looked at risk factors for intraparenchymal hemorrhage progression in his study of 113 head trauma patients with 229 acute IPHs. In this retrospective study, 4% of bleeds were smaller, 58% were unchanged, and 38% were bigger. Progression of IPH growth was independently associated with the presence of SAH, SOH, and the initial size of the parenchymal hemorrhage, with the strongest predictor of IPH progression being the presence of SAH[5]. In his study, the absence of growth of a hemorrhage on the second CT scan predicted a lack of further growth, and he concluded that in most cases, 1PH growth is seen early in the post-injury phase. He further recognized that factors strongly associated with surgical intervention were worsening Glasgow Coma Scale {GCS), significant IPH growth (> $5cm^3$), and effacement of cisterns on the initial CT scan**.**

Two retrospective studies by Chao and Figg look at the utility and efficacy of routine or serial CT scans in head injured patients. Chap's study looked at the utility of routine CT to predict the need for surgical intervention in blunt head injured patients. Sixty four of 198 patients in the study (323%) had worsening hemorrhages on their follow-up CT scans but only those with a worsening neurological exam had an invasive procedure as a result[6], He concluded that a routine serial CT head in a patient with an unchanged or normal neurological exam would not lead to invasive neurological intervention. Figg also looked at whether serial CTs would prompt surgical intervention in patients with GCS 8 with severe head injury and an intracranial pressure monitor. His results showed that no urgent surgical intervention was performed based on serial CT

scans in severe head injured patients who were non- surgical candidates based an the initial and repeat CT scans'. He also took into account the cost of performing these serial CT scans as they relate to radiologist charges, cost of nursing and transport personnel time, and the actual cost of the CT: the cost per CT was $655 per scan.

Carlos and Sifri both performed prospective studies on the topic of repeat CT scans in brain injured patients. Carlos looked at 100 patients in a 9 month period who had SAH, IPH, SETH, EDH, and contusions, of these patients, 68 (68%) underwent 90 repeat CTs. Routine scans were done on 90% of the patients; that is they had no change in the neurological exam which prompted the repeat scan, and 10% had a repeat CT scan after a change mental status or the clinical exam. Of the 90% who underwent routine scan without a clinical change, 26% of the scans were better, 51% were the same, and 23% were worse; no neurological intervention was performed based on these scans. Of the 10% who had a clinical change, 22% of the scans were better, II% unchanged, and 61% were worse. The conclusion of this prospective study is that if there is no change in the patient's neurological exam, the use of a repeat Cr is not supported[8]. Sifri's study looked at the value of a repeat CT scan in patients that had minimal he ad injury who had a normal exam. All patients in this study had a GCS of 5 13 with post-traumatic amnesia or loss of consciousness. 130 patients meet criteria and had a repeat CT within 24 hours of admission. At the time of repeat CT scan 76% of patients had a normal neurological exam and based on the repeat scan, none of the patients required a change in management or neurosurgical intervention. Also, none had a subsequent delayed neurological deterioration for the remainder of their hospital stay. Thirty one (24%) of patients had an abnormal neurological exam at the time of the repeat scan and 2 patients (6%) required neurosurgical intervention as a result (both had an acute change in mental status in the Emergency Department 2-3 hours after the initial CT scan). Rased on this prospective study, Sifri concluded that repeat CT's on patients with minimal head injury and a normal exam is not indicated.

In comparing the results of this study with the results of the aforementioned studies the risk of increasing hemorrhage falls in the 20% - 30% range. When comparing this study to Oertel's study, we have similar results in that contusions increase in size about 50% of the time, but in his study, 22% of EMI increased in size compared to my 33% and his SDH increased in size compared to my 4.5%. This could be a factor of study size, his being 142 patients to my 43. Also, in Ortel's study, the basis of hemorrhage size was based on the Neuroradiologist reading, and there may be discrepancy between what the Neuroradiologist sees as a stable or progressive scan as is evident in my study where 4 scans were read a stable, but if measurements were actually taken, there is proof that the bleed is in fact bigger. Another factor that, in all these studies, may alter the results is that CT scans are usually done in 5rnm slices, and that repeat CTs hardly ever reproduce the same cut us seen on the previous scan. This of course can lead to false reading of the size of a hemorrhage.

CONCLUSION

The use of CT scans as a tool to diagnose head trauma is paramount, but in this current health care environment, the need for conserving medical costs is becoming a great issue. Prospective studies have been done to show that serial CT scans on patients with minimal head trauma a normal neurological exam are not indicated, yet repeat imaging is still being done routinely. From a review of the literature, the following conclusions can be drawn: if a patient has a normal neurological exam and there is no clinical change during his hospital stay, a repeat CT scan is not indicated, In severe head injured patients with intracranial pressure monitors, serial CT scans are not indicated, if however, there is a change in intracranial pressure, a CT is indicated. In terms of evaluating patients with IPH and determining which patients may have progression of their hemorrhage, factors which may predict worsening of the hemorrhage are male sex, older age, a short time from injury to initial CT, and a prolonged PTT. If a patient has an associated SAH, SDH, or large IPH on initial CT, these correlate with progression of IPH.

Future studies should be directed at evaluating the presence of anti-platelet/anti-coagulation factors to predict progression of ICH, and the effect of using reversing agents to halt hemorrhagic progression.

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Rotatory Subluxation: Experience from The Hospital for Sick Children Type of activity: Case series and literature review

Alexandra Beier, DO Section of Neurosurgery, St John Providence Health System, Southfield, MI, USA

Abstract

Purpose: Diagnosis and management of atlantoaxial rotatory subluxation (AARS) is challenging clue to variability in clinical presentation. Although several treatment modalities have been employed, there remains no consensus on the most appropriate treatment. We explore this issue in our nine-year series of AARS.

Methods: Records of patients diagnosed radiographically and clinically with AARS between May 2001 and March 2010 were retrospectively reviewed. 0140 patients identified, 24 patients were male and were on average 8.5 years old (range 15 months to 16 years). Etiologies of AARS included trauma, congenital abnormalities, juvenile rheumatoid arthritis, post-infectious, post-surgical, and cryptogenic. Four patients had dual etiology. Symptom duration was variable: 29 patients had symptoms for less than four weeks, five patients had symptoms between four weeks and three months, and six patients had symptoms for three months or more.

Results: Treatment with a cervical collar was sufficient in 21 patients. One patient failed collar management and reduced with halter traction. Seven patients underwent initial halter traction, however four of these patients progressed and required halo traction. Two patients were placed in a halo upon presentation due to the severity of rotatory subluxation; both required subsequent operative fusion. One patient required upfront decompression and fusion due to severe canal compromise and myelopathy. All patients requiring operative fusion presented subacutely.

Conclusion: Management of AARS is variable due to the spectrum of clinical presentation. Those presenting acutely without neurological deficit can likely be managed in a collar; those who are irreducible or present with neurological deficit may require traction and/or surgical fixation. Patients presenting subacutely may be more prone to requiring operative intervention

Resident role: development of study, collection of data, analysis of data, primary author.

Rotatory Subluxation: experience from The Hospital for Sick Children

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Introduction

Atlantoaxial rotatory subluxation (AARS) occurs when the primary joint of rotation becomes disrupted and the atlas rotates out of synch to the axis. AARS can be seen with traumatic events, upper respiratory infections, congenital conditions associated with ligamentous laxity or vertebral anomalies, and can also occur spontaneously.[3-6,10] Although current treatment strategies include observation, traction, halo placement and operative fusion, there is no consensus on the best management approach. Some advocate operative fusion for persistent, chronic or recurrent cases of AARS, also termed atlantoaxial rotatory fixation (AARF) by some authors.[2,1o,13] Others advocate that long-term traction can be successful for chronic or recurrent AARF. There appears to be a trend toward conservative management for acute injuries presenting in less than three weeks, and operative fusion for chronic injuries that present greater than three months after the onset of symptoms. Controversy exists, however, for those patients presenting between three and twelve weeks.[1,6,13]Minimizing the number of operative fusions would be ideal, however prolonged symptoms, the inability to achieve reduction, and recurrence of AARS have been indications for fusion, lending to an operative fusion rate of approximately 30%.[6,13]We present a nine-year series of patients diagnosed and treated for atlantoaxial rotatory subluxation at The Hospital for Sick Children, spurred by our observation that our fusion rate is much lower than commonly reported.

Methods

In compliance with the Research Ethics Board at The Hospital for Sick Children, we reviewed medical records of patients who were diagnosed with AARS and treated by the department of neurosurgery at The Hospital for Sick Children from May 2001-March 2010. Patients had to be younger than 18 years of age at presentation, have a radiographic and clinical diagnosis of AARS, and had to be managed by the neurosurgery service. Exclusion criteria included radiographic diagnosis without clinical symptomatology and sole management by other departments.

Results

A total of 40 patients, 24 of them male, met our inclusion criteria. The average age at presentation was 8.5 years (range 15 months - 16 years). Etiologies for rotatory

subluxation included: trauma (n=20), congenital abnormalities (8), juvenile rheumatoid arthritis (1), post-infectious (7), post-surgical (2), and cryptogenic (6); four patients had dual etiologies. The most common presenting symptom was neck pain with limited range of motion (n=35). Other presenting symptoms included limited range of motion only (2), cranial nerve palsy (1), ataxia (1) and respiratory failure (1). Twenty-nine patients had symptoms for less than four weeks before presentation. Five patients had symptoms between four weeks and three months, and six patients had symptoms for three months or more. The average duration of symptoms prior to presentation was approximately three months, with a median of three days.

With regards to initial management, 22 patients were treated in a collar alone for a range of one day to seven weeks. Only one patient failed management after three weeks in a collar; this patient subsequently reduced with halter traction. Seven patients were initially treated with home nocturnal halter traction of 1-21 days duration. The weight used varied from 3-91bs. Three successfully reduced and were then followed in a collar for three to eight weeks. The other four patients progressed while in halter traction (range one day to one month); halo traction was successful in achieving reduction in this subgroup. Two patients were placed in a halo upon presentation due to the severity of their rotatory subluxation. One of these patients experienced continued subluxation upon ambulation despite halo immobilization and therefore underwent operative fusion. This patient was subsequently found to have juvenile arthritis. The second patient received halo traction followed by decompression and fusion due to marked rotatory subluxation and canal compromise. In one patient, surgical decompression and fusion was performed upon initial presentation due to severe spinal canal compromise associated with a one to two month history of motor regression and increasing ataxia. Finally, eight patients were observed without a collar or traction. The goal of treatment in all patients was improvement of pain and reduction of subluxation. This was obtained in 37 patients, whereas in three patients the rotatory subluxation appeared to be longstanding, fixed and without instability.

Discussion

The atlantoaxial (AA) joint can account for up to 60% of the neck's total rotation. This occurs due to the support of two main ligaments: the transverse ligament that prevents the forward subluxation of the atlas on the axis during head flexion and the alar ligaments that prevent excessive rotation.[2,7,11] Authors have found that at approximately 65 degrees of rotation, the spinal canal at the atlas can be reduced to approximately 7 mm in diameter, due to complete bilateral dislocation of the articular processes, causing severe cord compromise. With rotation of 45 degrees and 5 mm anterior subluxation of the atlas on the axis, the cord can be narrowed to 12 mm, also causing cord compromise.[2] Additionally, Villas et al have shown that atlantoaxial rotation as little as 36 degrees,

which is probably within the normal range of motion, can be associated with excessive contact loss of the articulating facets of C1 and C2.[14]
The immature pediatric cervical spine, especially the AA region, is subjected to higher torque and sheer forces that make this region more susceptible to injury with resultant instability.[12] Several factors predispose this region to injury including underdevelopment of spinous processes, horizontally oriented facet joints, weak neck musculature, immature bone ossification, a larger head to body mass ratio, and ligamentous laxity. In addition, the fulcrum of motion in the pediatric spine is located at the C2-3 level, much higher than in adults where it is located in the lower cervical spine.[6,12,13]

Rotatory deformities of the AA joint are typically transient and easily managed, However on occasion, they can be resistant to treatment and cause torticollis. Such persistent rotation was termed rotary fixation of the AA joint by Wortzman and Dewar in 1968.[15] The preferred term by Fielding was rotatory fixation since the fixation of the atlas on the axis may occur with subluxation, dislocation, or when the relative positions of the atlas and axis are still within the normal range of rotation.[2] The current literature unfortunately comprises a variety of definitions and terminology for this entity ranging from persistent rotation to pathological "stickiness".[2,8] Of note, many initial definitions and classifications were based on older technology including open mouth radiographs, lateral radiographs, and cineroentgenography.[2] Pang et al simplified the definition by classifying atlantoaxial rotatory fixation (AARF) as "inclusive of all gross departures from the normal rotational relationship between the atlas and axis. Further classification of the subtypes of AARF can then be predicted on a finer differentiation of the 'degree of pathological stickiness' ".[8]

A number of published reports have evaluated treatment strategies for AARS. Fielding initially presented his series of patients from which the classification arose in 1977. He reported on 17 patients, average age of 20.6 years, who had an average delay in diagnosis of 11.6 months. Surgical arthrodesis was performed in 13/17 (76%) patients. Fielding proposed that if a patient has rotatory fixation, the stability of the atlantoaxial joint might be compromised and therefore recommended traction initially to reduce the subluxation, followed by immobilization in a Minerva jacket for three months. They found that the risk of recurrence was greater with longstanding fixation of greater than three months and this was best treated with surgical fusion.[2] This has since been echoed by several studies.[5,10,13]

In 1989, Phillips and Hensinger presented their series of 23 children with rotatory subluxation. All children were hospitalized for halter traction. Halo traction was used when halter traction was ineffective. Patients were placed in a cervical collar once reduction had been achieved. Surgical stabilization was used for persistent deformity in

three children (13%). The recommended management strategies were based on symptom duration: for those with less than one week duration, a soft collar and bed rest for one week was implemented; patients were hospitalized for traction if reduction was not achieved. Patients with symptoms lasting between one week and one month were hospitalized for traction followed by post-reduction immobilization for four to six weeks. Symptom duration of greater than one month led to traction, attempted for up to three weeks, followed by arthrodesis in symptomatic patients who did not achieve successful reduction."

Subach et al in 1993 evaluated 20 children with AARS. Five patients were managed in a collar with adjunctive nonsteroidal anti-infiammatories. Halter traction was utilized in 15 patients. Nonoperative management failed in six patients (30%) requiring fusion due to recurrence or unsuccessful reduction.[13] In contrast to other reports, their study did not include congenital spine abnormalities, patients with Down syndrome or juvenile rheumatoid arthritis. Additionally, halo immobilization was not utilized.

Pang et al in 2005 presented their series of AARF. Their 35 patients were classified according to their proprietary classification system. Patients were deemed to either have AARF or be in a diagnostic grey zone (DGZ) after undergoing three full cervical computed tomography (CT) scans. All AARF patients were subject to traction regardless of duration. Halter traction was initiated for patients with symptoms less than three months, with reduction maintained in a Guilford brace. Cranial traction was used for patients with symptoms greater than three months duration or for patients who failed halter traction. Traction was not maintained beyond 2.5 weeks. These patients were immobilized in a halo vest after reduction. For patients falling into the DGZ, a soft collar, analgesics and muscle relaxants were used for two weeks. If this was unsuccessful, halter traction was initiated. Of 32 patients, six (19%) underwent surgical fusion.[9]

Our study found that the average length of symptom duration prior to presentation was approximately three months, with a median of three days. This skew suggested that this population could be pathophysiologically divided into distinct groups: an acute group (symptom duration of less than four weeks), a subacute group (symptom duration between four weeks and three months), and a chronic group (symptom duration of three months or greater). (Table 1)

Analysis of the chronic patients (N=6) revealed that three patients did not require active intervention and were therefore observed. These patients all had minimally symptomatic, non-progressing, and fused RS. Another patient in this group had a mild head tilt for three years with very mild limitation of rotation; this patient was also observed. The other two chronic patients presented after previous laryngeal infections. Both were

initially treated with halter traction, followed by halo traction and vest placement, and finally a collar, which was subsequently weaned. Of note, none of the patients in this subgroup required operative fusion.

In the subacute group (N=5), three patients underwent operative fusion. As previously mentioned, one patient presented with motor regression with accompanying severe cord compression, whereas the second was diagnosed with juvenile arthritis and had continued subluxation after halo placement. The third patient had underlying cerebral palsy and presented with pneumonia leading to respiratory failure. Investigation revealed severe cord compression. The patient was placed in halo traction followed by operative decompression and fusion. The other two patients in this group were treated with halter traction followed by a collar. One of these two patients recurred after being immobilized in the collar for one month and required halo traction for reduction. A halo vest was subsequently used for three months, followed by a collar for eight weeks.

In the acute patient group (N=29), no patient required operative fusion. Three patients were placed in halter traction. Of these three, one did not reduce, requiring halo traction and vest placement. The other two patients managed in halter traction were successfully transitioned to a collar. Of 22 patients initially managed in a collar, only one patient progressed requiring halter traction. Four patients were observed.

In our series, all of the patients requiring operative fusion presented subacutely. Historically patients in this subgroup appear to have been most variably managed. This group likely comprises patients with progressive etiologies that, in the absence of an inciting event, do not present to medical attention until significant subluxation and neural element damage has occurred. Their underlying pathophysiology makes non-operative management unsuccessful, and their monitored progression prompts surgeons to operatively fuse in an attempt to prevent further neurological compromise. The patients in this group should be closely observed for this reason. This is in contrast to the acutely presenting patients who often incur a traumatic event and present early in the course of their disease. Likely these patients have less derangement of normal anatomical supports, and as such are more easily maintained in a cervical collar. Interestingly, we did not observe the typical need for operative intervention in the chronic subgroup of patients. There may be several reasons for this, the most obvious of which is that only six patients presented chronically. Additionally, this group seems to represent unique pathophysiological entities, comprising such slowly developing conditions that autofusion and stabilization occurs by the time they present for evaluation. Operative fixation in these patients cannot be justified. Patients with laryngeal

infections likely have a normal anatomical substrate and therefore respond to non-operative forms of management.

Finally, only three patients (7.5%) underwent operative fusion, a figure significantly less than previous studies have reported. Several reasons may be implicated. First, the diagnosis was much less delayed overall than in previous reports.[2,9,13] As mentioned above, this is due to the majority of patients presenting acutely. Additionally, two previously published series excluded patients with congenital bony abnormalities, Down syndrome and juvenile rheumatoid arthritis, all of which typically increase the rate for surgical intervention.[9,13] Of our patients that underwent fusion, one had Down syndrome, one was diagnosed with juvenile rheumatoid arthritis and one had cerebral palsy. As such, the fusion rate in our series would have been essentially zero had we excluded these patients.

We must acknowledge the limitations inherent in the retrospective nature of this study. Recommendations for management strategies are difficult to develop based on review of treatment decisions made by treating surgeons, as these are often based on specific clinical scenarios and personal preference. It was also difficult to assess exactly how rotatory subluxation was radiographically diagnosed, as there were no standardized criteria for evaluation.

Conclusion

We present our recent experience in managing AARS. We operatively fused a lower proportion of patients than previously reported, and believe that the majority of patients will reduce and can be stabilized in a non-operative manner. All three patients requiring operative fusion presented subacutely; these patients likely represent a distinct pathophysiological group in whom thorough investigation and close monitoring is required.

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Treating chronic subdural hematomas early with craniotomy when twist-drill craniostomy with closed system drainage fails

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ABSTRACT:

OBJECTIVE: To examine the clinical outcome, hospital stay duration and recurrence rate of patients with chronic subdural hematoma (CSDH) that have initially undergone either twist-drill craniostomy (TDC) with closed system drainage, TDC with closed system drainage followed by craniotomy, or stand-alone craniotomy.

METHODS: Charts of 83 adult patients treated with either TDC with closed system drainage, TDC with closed system drainage followed by craniotomy, or stand alone craniotomy were retrospectively reviewed. The patients' clinical status at presentation and at discharge was evaluated using the Markwalder's neurological grading system, also recorded for all three groups was the intensive care unit and total hospital stay, and recurrence rates of hematoma.

RESULTS: All three groups (TDC, TDC followed by craniotomy, and craniotomy only) demonstrated significant and equivalent clinical improvement as assessed on the Markwalder's scale ($t(120) = 14.38$, $p < 0.0005$; $t(20) = 6.82$, $p < 0.005$; and $t(14) = 4.67$, $p = 0.0002$; respectively). In examining the overall length of stay in the hospital, the TDC group spent significantly fewer days in the hospital than did either the TDC plus craniotomy or craniotomy only groups (7.37, 11.9, and 13.27 days, respectively; for TDC vs. TDC plus craniotomy, $t(74) = 2.124$, $p = 0.037$; for TDC vs. craniotomy, $t(74) = 2.371$, $p = 0.020$). We observed that fewer days between TDC and craniotomy (< 2 days) led to significantly fewer days in ICU (6.5 days vs. 9 days, respectively; $t(6) = -5$, $p = 0.02$) and significantly fewer total days in the hospital (9 days vs. 15.5 days, respectively; $t(6) = -13$, $p = 0.003$). Further, there is significantly greater odds (83%) of an uneventful hospital course ($\leq$ 7 days in the ICU or $\leq$ 10 days in the hospital) if the interval between TDC and craniotomy is maintained at $\leq$ 2 days. The recurrences of bleeding was calculated to be 5% in TDC only, 18% in TDC followed by craniotomy and 22% in craniotomy only group.

CONCLUSION: Our data suggest that, when TDC with close system drainage fail and craniotomy is considered as a second tier treatment for CSDH, there is an 83% greater odds of an uneventful hospital course if the interval between TDC and craniotomy is maintained at $\leq$ 2 days.

KEY WORDS: Chronic subdural hematoma, Twist-drill craniostomy with closed system drainage, craniotomy

INTRODUCTION

Chronic subdural hematoma (CSDH), generally more prevalent in the elderly, is one of the most frequently encountered disease states by neurosurgeons. In the general population, the

incidence of CSDH is five per 100,000 per year.[33] However, among the geriatric population, the incidence rate has been reported to be fifty-eight per 100,000 per year.[17] By 2040, one-fifth of the total North American population is projected to be over the age of sixty five.[14] Thus, the incidence of CSDH is expected to significantly increase.

Surgical management of CSDH requires evacuation of a hematoma for direct decompression of the underlying brain in hope of a rapid clinical improvement. Although, many modalities have been reported in literature to evacuate CSDH and decompress the brain ranging from twist drill craniostomy to shunting to craniotomy, none have shown to improve postoperative outcome in the past 20 years.[43]

In 2003, Weigel et al[43] performed an evidence-based review of outcome of contemporary surgery for CSDH. Twist-drill craniostomy (TDC), burr hole craniostomy (BHC) and craniotomy were identified as the three principal techniques for surgical management of CSDH. TDC is generally a bedside procedure with a hand-held manual drill to make a burr hole with diameter less than five millimeters. BHC involves making burr holes ranging from 5 to 30 millimeters and is usually performed in the operating room. Finally, any surgical openings larger than thirty millimeters are considered craniotomies. The group concluded that all three techniques had similar mortality (2-4%). The TDC and BHC are safer than craniotomy and can be considered as first tier treatment and craniotomy can be considered as second tier treatment for CSDH.

Many of these elderly patients have multiple comorbid medical conditions, and thus there is a need for effective and minimally surgical intervention. Hence, TDC may be considered the initial procedure of choice. Smely et al[36] showed TDC to be a superior technique compared to BHC in terms of lowering morbidity, recurrence rate and duration of hospital stay. In 2003, Weigel et al[43] identified BHC to have better cure to complication ratio than TDC. There is demonstrable disparity in the reports from the literature. There is concordance, however in that CSDH patients treated with TDC, a bedside procedure, can often avoid more invasive procedures and the associated risks of general anesthesia.[6, 30]

To our knowledge, no clinical data has been reported that evaluates a definitive timing for following a first-tier treatment (TDC) of CSDH with a second-tier treatment (craniotomy). Our study examines the clinical outcome, hospital stay, and recurrences rate of CSDH patients that have undergone TDC, TDC followed by craniotomy, or craniotomy.

METHOD

Patients

This retrospective study reviewed charts of 83 adult patients (≥18 years old) who had undergone surgical treatment for CSDH evacuation with approval of the institutional review board. All patients had undergone TDC, craniotomy, or TDC followed by craniotomy between May 2007 and February 2011 at our institution. The coding in the medical records identified the cases. Patients found to have subdural collections of xanthochromic cerebrospinal fluid consistent with hygroma were excluded from this study.

Clinical Evaluation

Clinical information was obtained from hospital charts including admission notes, operative reports, and discharge summaries. Patient's age, sex, clinical features at presentation and at discharge, date of admission, days in intensive care unit (ICU) and ward, days with drain in place, and date of discharge/death were recorded. The patient's clinical presentation at admission and discharge was evaluated using the Markwalder's neurologic grading system for CSDH.[21] (Table 1)

TABLE 1. Markwalder's Neurologic Grading System[21]

Grade	State of Patient
0	Patient neurologically normal
1	Patient alert and oriented; mild symptoms, such as headache, absent or mild symptoms or neurological deficit, such as reflex asymmetry
2	Patient drowsy or disoriented with variable neurological deficit, such as hemiparesis
3	Patient stuporous but responding appropriately to noxious stimuli; severe focal signs, such as hemiparesis
4	Patient comatose with absent motor response to painful stimuli; decerebrate or decorticate posturing

Surgical Technique

At our institution, almost all patient with radiographic evidence of CSDH and Markwalder's grade greater than one underwent initially TDC with closed system drainage, or craniotomy at the discretion of the attending neurosurgeon based on the radiographic findings (high suspicion of blood being distributed between multiple septations) and clinical presentation. In the TDC with closed system drainage group, Integra™ subdural evacuation system was used with a 5mm drill bit to place the subdural drain using the standard technique.[2, 8] If the TDC with closed system drainage failed to achieve resolution of the mass effect or the thickest region of the CSDH was measured to be ≥ 1cm after TDC, patients underwent craniotomy.

Statistical Analysis

To establish that there were statistical differences among treatment groups, we used one-way ANOVAs to compare days in ICU and total number of days as inpatient. After the omnibus test, all pairwise comparisons were analyzed using contrasts stratified on treatment group (TDC, TDC followed by craniotomy, craniotomy). A similar analysis was run for comparison of Markwalder's grade, however, this was analyzed with repeated measures ANOVA. Due to the limitation in sample size of TDC patients who also received craniotomy, the analysis for the subsequent clinical course and outcomes of these patients was done using likelihood ratios. We defined an uneventful ICU course as ≤ 7 days in ICU, and an uneventful hospital course as ≤ 10

days total as an inpatient.[1] Conversely, eventful courses were considered any duration longer than those. All tabulation and chart making was done in Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) and all statistical analysis was done in SPSS (IBM Corporation, Somers, NY, USA).

RESULTS:

A total of 83 patient charts (69 males, 14 females) who had undergone treatment for CSDH were reviewed (Table 2). The mean age of the patient population was 70.6, ranging between 18 and 95 years old. We identified 63 patients who underwent TDC, 11 patients had TDC followed by craniotomy, and 9 patients were treated with craniotomy only. Due to the unequal number of patients between treatment groups, analysis of Markwalder's grade was conducted assuming non-homogeneity of variance.

Table 2. Basic descriptive features of the patient population analyzed

Variable	Number
Total number of patients	83
Gender	
Male	69
Female	14
Age	
Mean	70.6
	18 – 95
Treatment	
TDC[a]	63
TDC + Craniotomy	11
Craniotomy	9

[a]TDC, twist-drill craniostomy

Initial presentation and clinical status at discharge of CSDH patients was evaluated using the Markwalder's neurologic grading system for CSDH. All treatment groups illustrated mark improvement between initial presentation and clinical status at discharge in Figure 1. While no condition reached statistical significance, there is a clear trend that patients who received TDC had lower pre-treatment Markwalder's grade but also had better post-treatment Markwalder's grade. All three groups (TDC, TDC followed by craniotomy, and craniotomy only) demonstrated significant and equivalent clinical improvement as assessed on the Markwalder's grade ($t(120) = 14.38$, $p < 0.0005$; $t(20) = 6.82$, $p < 0.005$; and $t(14) = 4.67$, $p = 0.0002$; respectively). There were total of four mortalities observed, two in the TDC group (3.2%), one in the TDC followed by craniotomy (9.1%) and one in craniotomy only group (11.1%).

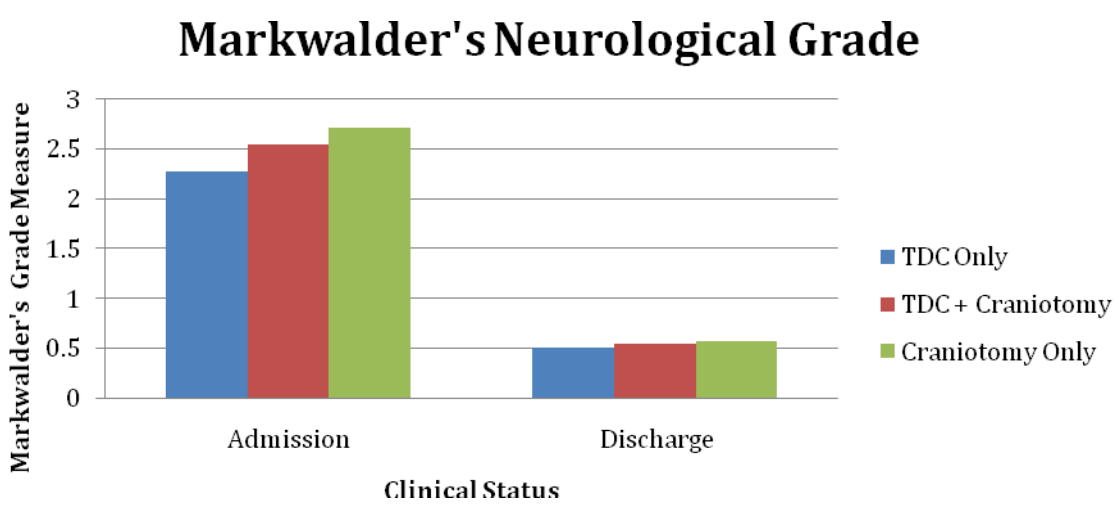

FIGURE 1. *Clinical evaluation of CSDH patients at initial presentation and at discharge after undergone TDC, TDC followed by craniotomy, or craniotomy only. Significant improvement in all three-treatment groups is noted.* TDC, *twist-drill craniostomy.*

There was a significant difference in length of ICU stay between patients in the TDC group compared to those in the TDC plus craniotomy (4.62 days and 9.20 days respectively; $t(74) = 4.955$, $p < 0.0005$) (Fig. 2). Furthermore, the length of ICU stay was significantly more in the TDC plus craniotomy group compared to craniotomy only (9.20 days and 4.14 days respectively; $t(74) = 3.79$, $p < 0.0005$). However, no significant difference in length of ICU stay between TDC only and craniotomy only groups was found (4.62 days and 4.14 days respectively; $t(74) = -0.438$, $p = 0.663$). In examining the overall length of stay in the hospital, the TDC group spent significantly fewer days in the hospital than did either the TDC plus craniotomy or craniotomy only groups (7.37, 11.9, and 13.27 days, respectively; for TDC vs. TDC plus craniotomy, $t(74) = 2.124$, $p = 0.037$; for TDC vs. craniotomy, $t(74) = 2.371$, $p = 0.020$). However, the duration of hospital days between the TDC plus craniotomy group and craniotomy only group were not significantly different from one another ($t(74) = -0.46$, $p = 0.654$).

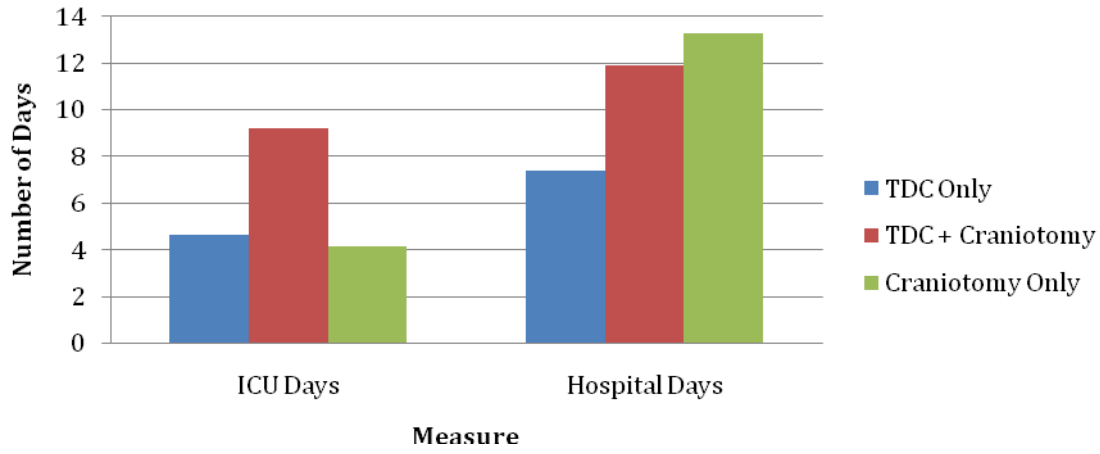

FIGURE 2. *Length of stay in ICU and total duration of hospitalization for three treatment groups. Note that the single-modality conditions (TDC only and craniotomy only) had similar and shorter ICU stays than the bimodal treatment (TDC+craniotomy) while TDC only had a much shorter total length of stay as an inpatient than either TDC+craniotomy or craniotomy only.* TDC, *twist-drill craniostomy*

Eleven patients received craniotomies (13.3% of our sample) after having undergone TDC with closed system drainage. In figure 3, we observed that fewer days between TDC and craniotomy ($\leq$ 2 days) led to significantly fewer days in ICU (6.5 days vs. 9 days, respectively; $t(6) = -5$, $p = 0.02$) and significantly fewer total days in the hospital (9 days vs. 15.5 days, respectively; $t(6) = -13$, $p = 0.003$). Furthermore, both days in ICU and total days in the hospital were tightly correlated with days intervening between TDC with closed-system drainage and subsequent craniotomy ($R = 0.89$ and 0.98, respectively)

Patients with a $\leq$ 2 day duration of TDC drainage had a likelihood of 0.66 for an uneventful ICU course. Comparatively, no patients who remained with TDC drainage for > 2 days duration had an uneventful ICU course. Therefore, there are 66% odds for a $\leq$ 2 day duration TDC course yielding an uneventful ICU course compared to a > 2 day duration of TDC. By the same token, the likelihood of $\leq$ 2 day TDC duration patients to have an uneventful total hospital stay was 0.83 while again, nobody in the > 2 day TDC drainage group met the threshold for an uneventful course. This suggests 83% greater odds of an uneventful hospital course if the interval between TDC and craniotomy is maintained at $\leq$ 2 days. Despite this obvious difference, there was no significant difference between the likelihood of any particular Markwalder's grade having an eventful or uneventful course. Among the patients who received craniotomy after TDC, those who received the craniotomy $\leq$ 2 days after the TDC placement had slightly better Markwalder's grade (mean = 0.5, range 0 – 1) than did those who received craniotomy > 2 days

after TDC placement (mean = 0.75, range 0 – 2) suggesting that worse grade patients are less likely to improve with a longer course of drainage after TDC.

Of our sample of 83 patients, seven patients had similar or worse symptomatic recollection of fluid after initial therapy than on initial presentation, defined as recurrence. Of these, three were in the TDC group; two were in the TDC and craniotomy group; and two were in the craniotomy only group. One patient in the TDC only group was readmitted because of rebleeding. Recalling that there were uneven sample sizes in the three groups, we considered these recurrences in terms of ratio to group size. 22% of craniotomy only patients, 18% of TDC and craniotomy patients, and 5% of TDC patients had recurrences of bleeding post-treatment (Fig. 4).

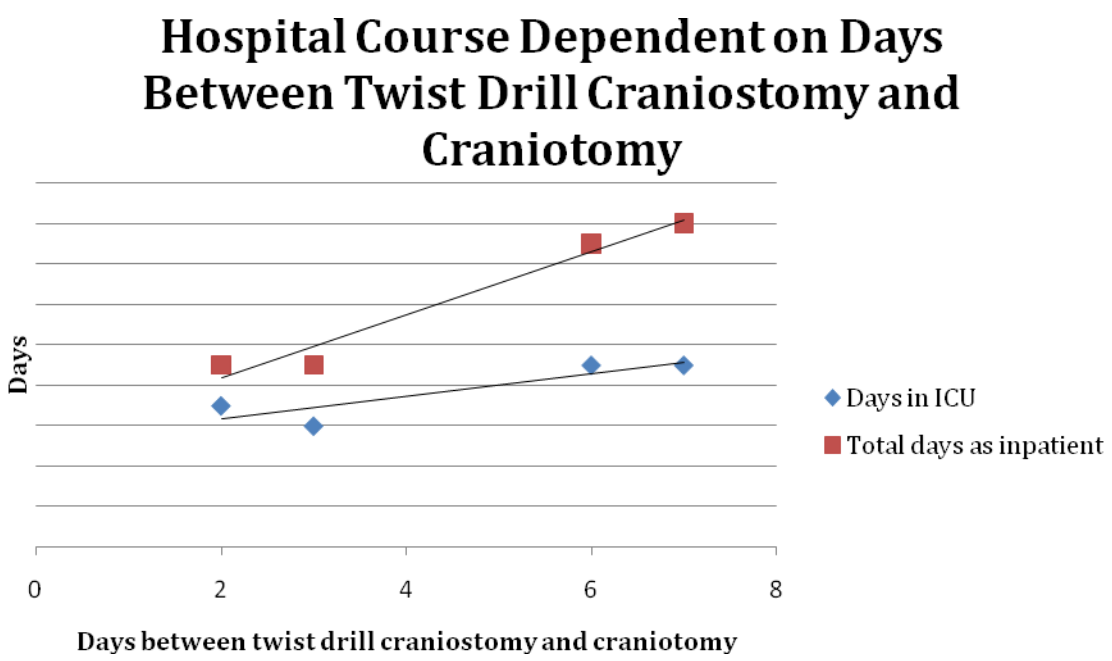

FIGURE 3. Trendlines showing the number of days post-intervention in ICU and total spent as inpatient stratified by number of intervening days between TDC followed by craniotomy. TDC, *twist-drill craniostomy*

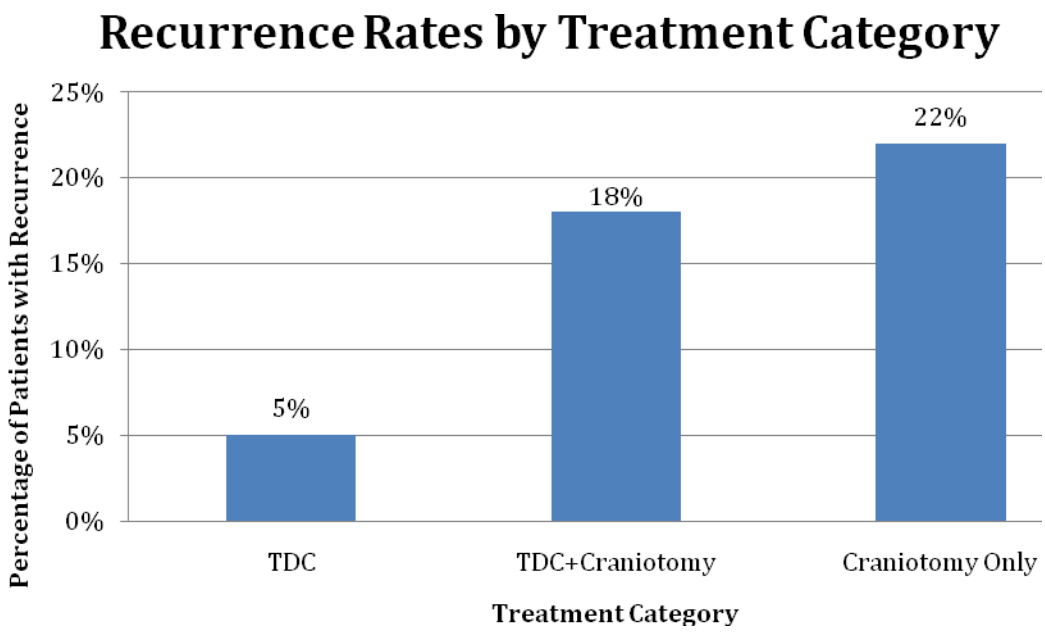

FIGURE 4. Rates of recurrence by treatment category (TDC, TDC+craniotomy, and crantiotomy. TDC, *twist-drill craniostomy*

DISCUSSION

Chronic subdural hematoma (CSDH) is prominently a disease of the elderly. The purpose of our study was to determine whether there was a definitive timing for following a first-tier treatment (twist-drill craniostomy; TDC) with a second-tier treatment (craniotomy). Data revealed that there is an 83% greater odds of an uneventful hospital course if the interval between TDC and craniotomy is maintained at $\leq$ 2 days (Fig. 3). Sindou et al[35] performed a prospective randomized study involving 65 patients treated with TDC with closed system drainage. They observed that a short duration of drainage (48hrs) is as effective as longer duration of drainage (96hrs). Our study supports the finding that if TDC with closed system drainage is not effective within the first forty-eight hours, any longer drainage may not be any more effective or efficient in terms of improving clinical outcome or reducing duration of hospitalization, respectively. We examined the clinical outcome, hospital stay, and recurrences rate of CSDH patients that had initially undergone TDC or craniotomy, and TDC followed by craniotomy.

First, clinical evaluation of these eighty-three patients surgically treated for CSDH revealed significant improvement in their Markwalder's neurological grade[21] from initial presentation to their discharge from the hospital in all three-treatment groups (Fig. 1). We observed 3.2% (2 patients) mortality rate in the TDC group, 9.1% (1 patient) TDC followed by craniotomy group, and 11.4% (1 patient) in craniotomy only group. Both mortalities in the TDC and TDC followed by craniotomy group were due to respiratory distress and acute myocardial

infarction on presentation, respectively. The other two patients, one in TDC followed by craniotomy group and one in craniotomy only group expired from cardiopulmonary arrest secondary to withdraw of care by family. Similarly, Weigel et al[43] reported in a meta-analysis of forty-eight suitable articles that the mortality rate in the TDC ranged from 0 - 7.9% and for craniotomy group ranged from 0 - 11%. However, the analysis was unable to reveal a significant difference in mortality between the three techniques. They identified a similar rate of effectiveness, recurrences, and complications between TDC with closed system drainage and open craniotomy.[35, 43] Furthermore, Ramachandran and Hegde[29] noted that the patient's age, initial clinical presentation, and associated illnesses (cardiac or renal failure) play a significant role in their mortality. Recently, Miranda et al[23] observed old age (≥ 65 years old) as the sole factor in predicting in-hospital mortality of CSDH patients. The type of intervention (TDC with closed-system drainage, burr-hole craniostomy (BHC), or craniotomy), radiographic findings, and anticoagulant agent use did not affect the mortality rate.

Second, no significant difference in intensive care unit (ICU) stay as well as total hospitalization was noted between the three treatment groups (Fig. 2). No significant difference in the ICU stay between the first tier-treatment, TDC and the second-tier treatment, craniotomy was noted. The ICU duration, for the patient who had undergone both TDC and craniotomy was much longer than the other treatment modalities; however the overall hospital stay between this group (TDC plus craniotomy) and craniotomy only was not significantly different ($p > 0.654$). Patient treated with TDC only were observed to have much shorter hospitalization than TDC plus craniotomy or craniotomy only group (7.37, 11.9, and 13.27 days, respectively; for TDC vs. TDC plus craniotomy). Therefore, performing TDC for patients with CSDH may be a better option than craniotomy if they do not require further treatment with craniotomy in terms of shorter hospital stay as well as less invasive procedure and avoiding risks of general anesthesia.[30, 31] In our study, the patients who underwent craniotomy did so at the discretion of the attending neurosurgeon based on the radiographic findings (high suspicion of blood being distributed between multiple septations) and high Markwalder's grade.

Although, considerable literature has been published regarding management of chronic subdural hematoma, the optimal treatment has not been established. BHC has been stated to be the most efficient choice for CSDH.[19, 43] Gelabert-Gonzalez et al[5] reviewed charts of one thousand CSDH patients that had undergone BHC with closed drainage and concluded that poor prognosis is related to the patient's age (>70 years old) and clinical grade on admission (grades 0-2 versus grades 3-4). Gokmen and colleagues[6], in a randomized prospective study of 70 patients, 38 patients were treated with TDC and 32 patients were treated with BHC, found both surgical techniques to be effective treatment options. However, TDC, a less invasive procedure done under local anesthesia had shorter operating time compared to BHC. In addition they also observed that the longer the hospitalization, the higher the overall mortality.[6]

Third, the reported recurrence rate for CSDH ranges from 2.3 to 33%.[3, 16, 24-26, 37, 39, 41, 43] In the literature it has been speculated that the hematoma itself is the promoter for its chronicity due to high concentrations of vasoactive cytokines, and inflammatory mediators and fibrinolytic

factors that may be involved with membrane formation and recollection of the hematoma, respectively.[4, 7, 9-13, 15, 18, 20, 22, 27, 28, 32, 38, 40, 42, 44] In our study, seven patients (three in TDC group, one in TDC followed by craniotomy and two in craniotomy only group) had recurrence after initial therapy. One of these patients in the TDC group expired. This patient was readmitted and underwent craniotomy; this patient, however decompensated and the family withdrew care. Because of the unequal group sizes in our data, we considered these recurrences in terms of ratio to group size (22% of craniotomy only patients, 18% of TDC and craniotomy patients, and 5% of TDC patients) had recurrences of bleeding post-treatment (Fig. 4). The recurrence rate found to be low in our TDC group is most likely due to closed-system drainage, which is consistent with the literature.[2, 34, 43]

The limitations of our study are its retrospective nature and the unequal and small number of patients in the TDC followed by craniotomy and craniotomy only groups. To attempt to account for the unequal group sizes, all statistical analyses were done assuming non-homogeneity of variance. Further, due to the limitation in sample size of TDC patients who also received craniotomy, the analysis for the subsequent clinical course and outcomes of these patients was done using likelihood ratios. TDC with closed-system drainage; a bedside procedure may be better option in the elderly CSDH patients because it is a minimally invasive technique, may be performed under local anesthesia and our results indicate that there is significantly greater odds (83%) of an uneventful hospital course if the interval between TDC and craniotomy is maintained at ≤ 2 days.

This may decrease the overall hospital stay that, in turn, may decrease the healthcare expenditure of admission for CSDH. While some of the time spent in ICU by the patients in our study can be accounted for simply by time due to the therapeutic interventions themselves, each group had ICU time associated with its own intervention types. However, patients receiving TDC and craniotomy may have had more time in the ICU because of the dual nature of the intervention they received. To clarify this confusion, the study will be repeated in the future with a larger sample size and more focused group assignments with equal numbers of patients in each group and the addition of a new group – TDC, TDC followed by craniotomy (≤ 2 days), TDC followed by craniotomy (> 2 days), and craniotomy only. Furthermore, a parallel study should be conducted that is prospective, large-scale and with randomized patient assignment, adhering, of course, to standard of care, such that these patients can be subject to pre-planned (rather than retrospective) measures and long-term follow-up to better identify the distinctions in outcomes and duration of hospital stay between these groups.

CONCLUSION

Our data confirms that TDC with closed system drainage is an effective and efficient first-tier treatment for elderly patients with CSDH by improving Markwalder's neurological grade and with shorter duration of hospitalization. However, when TDC with close system drainage fail and craniotomy is considered as a second-tier treatment for CSDH, our data

revealed that there is an 83% greater odds of an uneventful hospital course if the interval between TDC and craniotomy is maintained at $\leq$ 2 days.

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Unsual presentation of a progressive cervical myelopathy secondary to epidural venous engorgement

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Summary

The authors report a case of progressive spastic quadraparesis, numbness and gait ataxia in a 42 year old male; which was attributed to cervical epidural venous engorgement from C2-6. The patient has a known history of ventriculoperitoneal shunt placement from his childhood that was last revised in 1980. His workup initially revealed a ventral cystic mass on MRI extending from C2-6. Spinal angiograms did not reveal any AV malformation or fistula. After extensive review of the literature, it was felt that this was a progressive myelopathy from cervical cord compression secondary to the venous engorgement. We decided to treat the patient with revision of his VP shunt system and utilizing a programmable Medtronic strata reservoir siphon guard. His settings were sequentially changed until both radiological and clinical improvements were noted. The patient is now one year post surgical intervention, with resolution of his myelopathic symptoms.

Key words: cervical myelopathy, cervical plexus enlargement, intracranial hypotension

Introduction

Myelopathy is a relatively common disorder with a myriad of possible differential diagnosis. However, myelopathy as a result of cervical spinal cord compression from epidural venous engorgement is very rare condition with only two previously reported cases that had surgical treatment (see image 1). The epidural space is rich in vasculature; the cervical epidural veins form anastomoses with the suboccipital venous plexus, anterior condylar vein, and the vertebral arteriovenous plexus. However, dilation of the cervical epidural veins can occur in this region with an increased outflow of blood. A more common cause of this enlargement, as reported in other cases[1], is spinal vascular malformation, or expanded collateral pathway from thrombosis of the internal jugular vein[2,3].

Image 1:

Previously reported cases of myelopathic related to Intracranial Hypotension

Authors	Case presentation	Age, Sex	Time to presentation	Surgical Treatment	Clinical Outcomes
Miyazaki et al, 1998	gait ataxia with clumsiness, history of Post-	53, M	15 months	Shunt ligation	Improved

Wingerchuk et al, 2005	SAH hydrocephalus Weakness and gait instability, history of porencephalic cyst	18, F	16 years	Shunt revision	Improved
Junn et al, current case	Spastic quadraperisis, history of VP shunt after CVA at birth	42, M	30 years	Shunt revision	Improved

The Monroe Kellie doctrine has been used to explain the occurrence of cervical plexus enlargement in the face of intracranial hypotension[4]. The doctrine defines the intracranial total volume is constant and is the summation of CSF, blood, and brain volume. Since the brain volume is relatively constant, a drop in CSF volume, as occurs with intracranial hypotension, results in overall increase of the cranial blood volume. Although this primarily affects the dural sinus volume, this has been shown to apply to the spinal canal in previous studies[5]. Clinically, patients with intracranial hypotension typically present with postural headaches; although not always[6], as in our case report. Other clinical findings include nausea, vomiting, neck pain, blurred vision. A less common complaint includes radicular symptoms, as previously reported[7]. In our case, this 42 year old developed a progressive myelopathy secondary to excessive draining of CSF from his ventriculoperitoneal shunt. After revision of his shunt with a programmable Medtronic valve; the patient was treated by sequentially adjusting the flow setting on the shunt. He improved clinically, as did the radiological images.

Case report

History and Physical

In our case, a 42 year old male, left handed, that was previously shunted presented with a progressive spastic quadraparesis. He was initially shunted at 30 days of age subsequent to a cerebral infarct and resultant hydrocephalus. The patient did well post operatively, however he did require multiple revisions. The last revision was in 1980, approximately 30 years ago. Over a course of 4 months prior to his initial evaluation with our service, the patient began developing a spastic quadriplegia. He denied any headaches. On examination, the patient was found an ataxic gait; he could barely stand on his own, and needed assistance with taking steps. Cranial nerves II-XII were grossly intact. The patient did have long tract signs including a Hoffman, and Tromner. Dysdiadokinesia was present in the bilateral upper and lower extremities, although mild. Reflexes were symmetrical at ¼ in all four extremities. Manual muscle testing did reveal spasticity in all four extremeties, and strength of the left side at 3+/5 and right side at 4-/5. In

addition to this the patient did complain of radicular symptoms into his left upper extremity that followed a C5,6 dermatomal pattern.

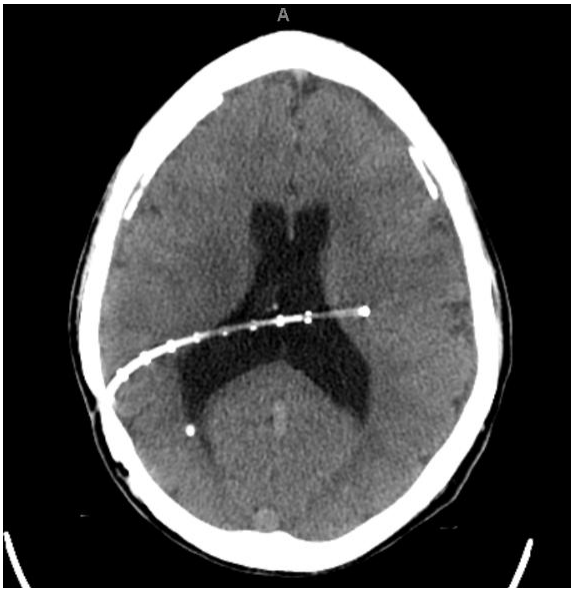

Image 2 – *Preop CT brain without contrast.*

Review of the head CT revealed that the shunt was in appropriate placement, and no acute cerebral events were seen on the study (see image 2). The patient's cervical MRI revealed a ventral cystic mass extending for C2-6 causing central canal stenosis (see Image 3). This mass enhanced on contrast images, and the referral was made for biopsy of this mass. After careful review, we decided to rule out an AVM or dural fistula. The patient was referred out for a spinal angiogram, which did not reveal any obvious lesion other than the cervical venous engorgement. A cervical myelogram was also performed which confirmed the cervical stenosis (see image 4).

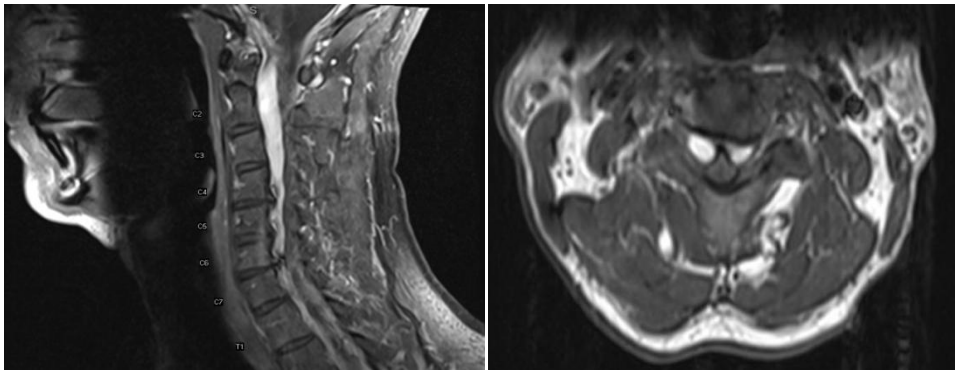

Image 3 – *Preop MRI of the cervical spine with contrast. On the left is a sagital cut showing the ventral enhancing cystic pocket extending from C2 to C6. On the right image the ventrally located venous engorgement is noted bilaterally, causing central canal stenosis*.

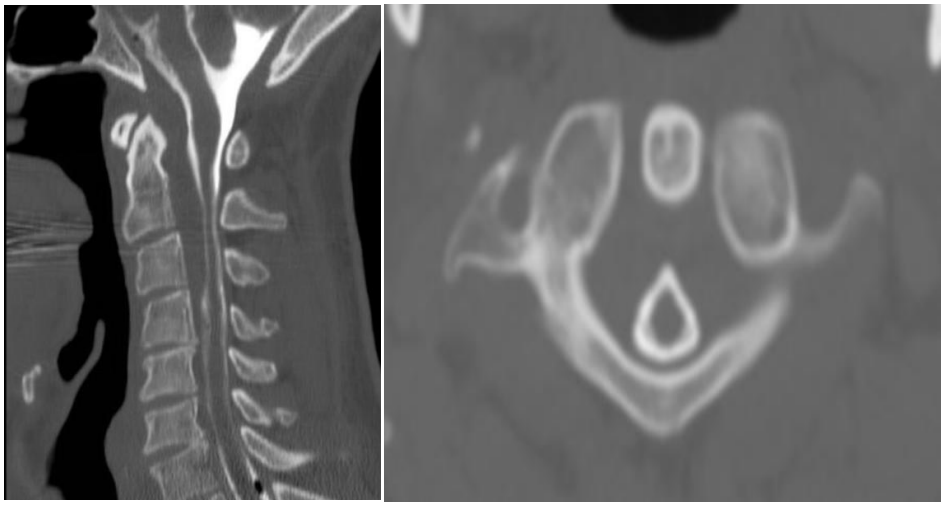

Image 4 – *Preoperative CT myelogram of the cervical spine. On the left, a midsagittal image reveals the severe stenosis caused by the ventral venous engorgement. On the right, an axial view is seen confirming the severe stenosis.*

Once the MRI, CT myelogram and spinal angiogram were performed it was felt that the finding was that of cervical venous engorgement secondary to over shunting from the current ventriculoperitoneal (VP) system. A review of the literature, helped confirm the diagnosis and decided to treat the patient with revision of his VP Shunt.

Treatment and Postoperative Care

Unlike the previously reported treatment plans, we decided to directly perform a VP shunt revision utilizing a Medtronic programmable shunt system, strata® valve with anti-siphon device (see image 5).

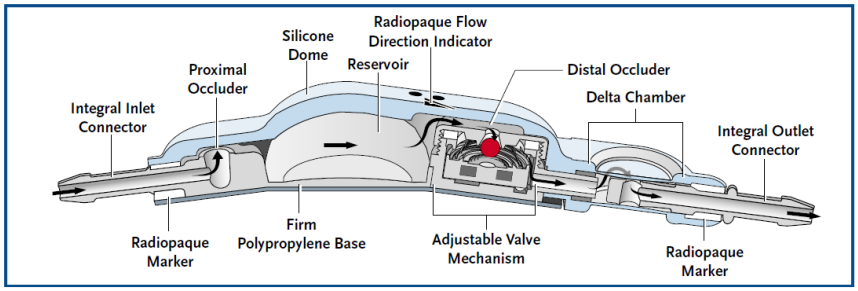

Image 5 – *Strata valve cutaway(used with permission)*

In previous reports, patients were initially had a lumbar drain placed and then the drain height was slowly raised to higher pressures. Once optimal pressures were met, then a shunt was placed with those pressures. In our case, the patient was taken to the OR and we tested intraoperatively the patency of both proximal and distal catheters. They were both found to be patent. Once, confirmed we them simply replaced the previous shunt reservoir with the programmable strata valve. The shunt was set at the lowest outflow setting (2.5) to allow for correction of the prior over shunting. At two weeks post operatively, the patient was brought into clinic for suture removal and a setting change to 2.0. At this point, the patient had noted some

improvement and was able to sit up from his wheelchair. The patient returned 4 weeks later, for revaluation and shunt setting change. He was able to walk into his appointment utilizing a walker. His shunt setting was changed at 1.5 at that point. At his next appointed, the patient was noted to near his baseline, with only occasionally utilizing a cane. His strength had improved to 5/5 on the right side and 5-/5 on the left side. A post op MRI of the cervical spine and CT of brain was ordered at this point (see images 6 and 7). At the one year follow up, the patient is found to be back at baseline and doing well.

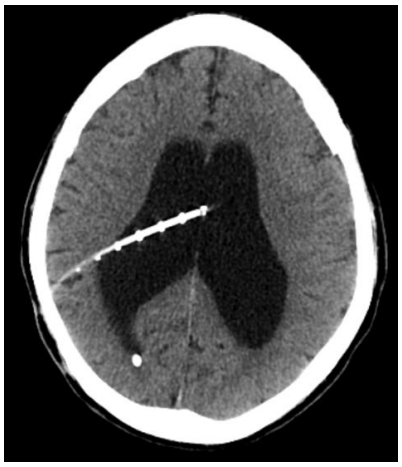

Image 6 – *CT of the brain without contrast taken 10 weeks post op. the ventricle have enlarged, without any significant effacement of the sulci*.

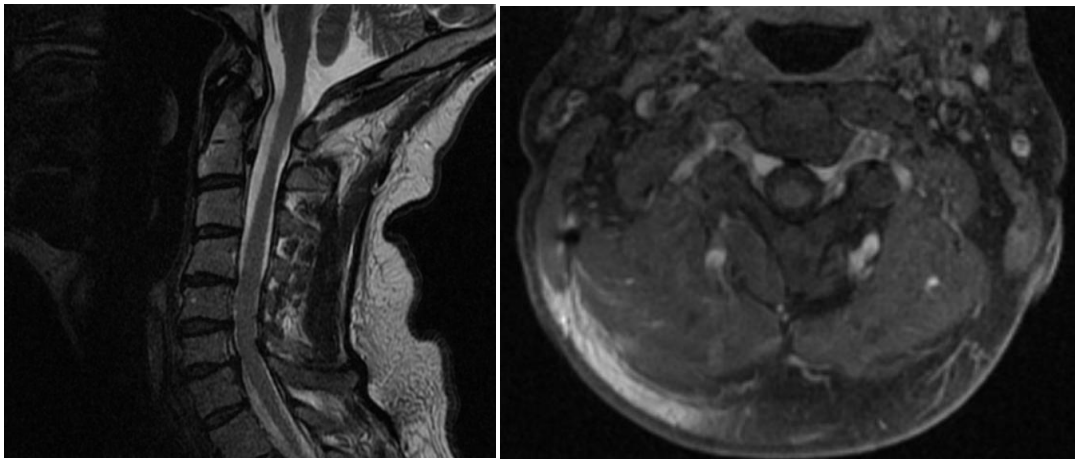

Image 7 – *MRI of the cervical spine, 10 weeks post operative. On the left, a T2 sagittal MRI of the C-spine reveals interval regression of the cervical venous engorgement. On the right, a T1 axial image with contrast shows the significant decrease in size of the anterior cervical veins and improvement of the cervical stenosis.*

Discussion

In summary, this patient's spastic quadraparesis was a result of cervical cord compression from engorged ventral cervical epidural veins. The etiology was determined to be from intracranial hypotension from VP over shunting. The mechanism is explained by the Monroe Kellie doctrine. As the CSF volume was decreased from the over shunting, the intracranial

blood volume increased. This then allowed for the increase in venous drainage that lead to the cervical epidural vein engorgement. To reverse the cervical venous engorgement, the intracranial hypotension was reversed by slowing down the CSF outflow. The programmable Strata valve was utilized and the shunt was set as the lowest outflow to allow for CSF fluid built up. As the ventricles grew in size again, the intracranial blood volume decreased and thereby decreasing the outflow. Over a 10 week period the setting were changed until there was resolution of the patient myelopathy. Post operative MRI of the cervical spine revealed the regression of the cervical vein engorgement. At one year follow up visit, the patient was still doing well with resolution of his spastic quadriplegia.

Our treatment plan differed from previously reported cases. We decided to bypass the use of a lumbar drain, in order to reduce the chances of infection, and other associated complications. The Medtronics programmable shunt valve was felt to be the amenable option, given its ease of use. We decided to use a slow approach in shunt setting changes, given the risk of causing subdural hematomas. Finally, given the wide differential for this ventral mass, it is highly recommended to obtain an MRI with and without contrast, as well as a formal spinal angiogram.

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Title: A Benign Outcome: Presentation of Intraparenchymal Cystic Lesion Containing Ectopic Choroid Plexus Tissue in a Female with Known Metastatic Breast Cancer: **Case Report**

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Abstract:

The authors describe a case of a 53 year old female who presented with slight difficulty with memory and ongoing headaches for approximately two months. She was previously treated for breast cancer with metastatic spread to the bone. Her oncologist requested an MRI of the brain which showed an Intraparanchymal, hyper intense, enhancing, infiltrative cystic lesion with mass effect in the right frontal lobe which had increased compared to a scan six years prior. Differential diagnosis suggested a ganglioglioma, metastatic disease or a low grade astrocytoma. She was advised to undergo surgical intervention for resection and biopsy of this mass. Intraoperative frozen specimens were suggestive of a possible meningioma vs metastatic disease; however, final pathology demonstrated ectopic choroid plexus tissue, a completely benign process. While a pleasant surprise for both the patient and the surgical team, the final diagnosis was rare and unusual.

A comprehensive review of the literature showed that benign intraparenchymal epithelium-lined, non communicating cysts incorporating ectopic choroid tissue (**ChP**) or ChP-like tissue has only been reported four times before, making this a 5th reported case.[8,10,12] However, other most reported cases were consistent with a malignant process such as Choroid Plexus Papilloma (**ChPP**) and one case of Choroid Plexus Carcinoma (**ChPC**).[1,2,4,10,12] Similarly, formation of ectopic ChP or ChP-like tissue in other areas of the body, outside the brain parenchyma, such as cerebellopontine angle, presacral area, sacral canal, intramedullary area, cervical region, face, and subpleural space have also been reported.[13-20] As previously questioned, the mystery of ectopic ChP tissue formation still remains, and according to Di Rocco, it is difficult to regard these cystic lesions as simply a homologous group of just one type of pathology.[8,10] Theories of primary or secondary ectopia cannot be excluded, with regards to ectopic ChP tissue presentation, outside of anatomic areas housing the choroid plexus. Similarly, the formation of the cystic cavity seen in our patient's lesion and its ultimate expansion can potentially be explained by direct production of CSF by the ectopic choroid plexus found within in the cystic mass.

Conclusion:

Even though choroid plexus tissue is mostly found within the areas of cerebral spinal fluid production and ventricular systems, ectopic growth can occur. Intraparenchymal, ectopic ChP tissue has been reported before, this being a 5th reported case to our knowledge, along with many other areas of the body. Our patient's case is unique to her past medical history and presentation. While pathologic evidence definitively excluded this as metastatic spread, and suggested more of a benign process, further evaluation of the specimen with additional

immunostains and markers may be of benefit, to definitively exclude a malignant process, based on other markers described in the literature.

Key words:

1) Ectopic choroid plexus
2) Intraparenchymal choroid plexus
3) Choroid plexus
4) Intraparenchymal
5) Cerebrospinal fluid
6) Cystic lesion

Introduction:

Choroid plexus (ChP) are papillary structures that are usually only found protruding in the ventricular cerebral spinal fluid (CSF) and ventricular system. They occur in each of the four major cisterns developing from median wall of each lateral ventricle and from the roof of the third and fourth ventricle. Microscopically all plexuses share a similar structure, consisting of an external simple cuboidal epithelium surrounding a vascular bed embedded in a loose connective tissue (**Figure. 1**). They are viewed as tight epithelium enclosing a ventricularized stroma, and as such form and interface between the blood and the cerebrospninal fluid (CSF). CSF production and secretion is the key feature of (ChP), however, ChPs role has been suggested in influence of brain development and maturation, neurohumoral modulation of the brain, component of neuroimmune system, and pharmaco-toxicological aspects of the CHP.[11]

According to the review by *Strazille and Chersi-Egea*, morphogenesis of the ChP is an early event in CNS development and occurs before the pontine flexure appears on the 5 vesicle-segmented neural tube. Times of earliest histological evidence of choroidal differentiation, as reviewed for several species by *Catala*, indicate that the metencephalic plexus (fourth ventricle) generally appears first, followed by the telencephalic plexuses (lateral ventricles), and finally the diencephelic plexus (third ventricle).[7]

During initial review of our patients imaging studies and a known history of breast cancer complicated by bone metastasis, differential diagnosis of the right frontal cystic mass were in favor of ganglioglima vs metastatic disease. However, final pathologic diagnosis post resection was consistent with a cystic lesion incorporating benign ChP tissue. A comprehensive review of the literature showed that benign intraparenchymal epithelium-lined, non communicating cysts incorporating ectopic ChP or ChP-like tissue has only been reported four times before, making this a 5th reported case.[1-6,10,12]

Case Report

History and Examination: This is a 53 year old female who presented to our office complaining of headaches. Headaches were described as gradual in onset but in a persistent pattern over a period of two months. Headaches were described as moderate and worse in the morning, especially at the time of awakening. Headaches were noted to dissipate throughout the day. Furthermore, she stated that in this period of time she has been having difficulty with short

term memory and ability to concentrate. Prior to her visit with the neurosurgeon, she consulted with her oncologist who recommended and MRI of the brain, which showed a right frontal mass.

She underwent a comprehensive neurological examination in the office which showed her to be completely neurologically intact, without any neuro-motor or neuro-sensory deficits. Her long term and short term mammary was also found to be intact along with her attention span.

Imaging studies: Subsequently imaging studies were reviewed. A PET Oncology study from 03/2010 using F-18-fluorodeoxyglucose isotope reviled abnormal uptake in mediastinum, inferior pubic ramus, left posterior rib cage, and left mandible all consistent with known metastatic disease. No abnormal uptake was noted in the brain or other soft tissue structures. MRI of brain with and without gadolinium was obtained on 6/2010 (3-T MRI unit) showed an enlarging high T2, low T1 signal cystic mass in the right frontal lobe, with hypo-intense rim, no diffusion restriction, with mild peripheral enhancement (**Figure 2B**). These imaging findings were seen on an MRI performed on 8/2003 (**Figure 2A**), however, the lesion has enlarged in that interval of time. An additional 1 cm round hyper-intense lesion was noted in the left parietal calvarium, which was new from 8/2003. Differential diagnosis included glioma, oligodendroglioma, and less likely metastatic disease.

Treatment options: Since the lesion has increased in size and given the patients history of breast cancer with metastatic spread to the bone she was offered treatment in form of surgical resection of the right frontal lesion with biopsy at that time. Given her ongoing history of morning headaches, and difficulties with memory and concentration, the right frontal mass was the likely cause. Patient agreed to surgical intervention on 8/13/10.

Operation: Patient underwent stereotactic neuronavigational assisted right frontal craniotomy and gross total removal of intraaxial mass. Intraoperatively the cystic cavity was opened and yellowish fluid as aspirated. Furthermore, within the cyst a calcified material in the cystic wall was encountered. Material of the cyst wall was sent for frozen section diagnosis, initially suggestive of possible meningioma versus metastasis. Subsequently a visual gross total resection was performed.

Postoperative Course: Patient tolerated procedure well, and was found to be neurologically intact post op. Patient was transferred out of ICU on post op day two and was doing well. Post-operative MRI was performed on 8/14/10 was reviewed by both neurosurgery and neuroradiology teams; residual cystic cavity was noted without any signs of residual enhancing tumor, with some minimal vasogenic edema for which patients was taking Decadron. She was asked to follow up in the office for final pathology review and further treatment planning.

Pathology interpretation: Gross specimen and fluid collected was reviewed. Interpretation of fluid FNA cytology was consistent with hypocellular hemorrhagic histiocytic contents of a cyst, without identification of any other cellular components. Surgical pathology showed cystic lesion within the cortical brain parenchyma with surrounding areas of gliosis and hemosiderin deposition. Cyst was lined by cuboidal benign appearing cell. Tissue found within the cyst and within the brain parenchyma, consisted of benign appearing cuboidal cells, resembling that of the cyst wall lining. There was abundant psammomatous calcification within and around the

epithelium and focal fibrovascular cords suggestive of papillary configuration, without any mitotic figures. Ki-67 stain demonstrated a very low proliferative grade. Given the patients history of breast cancer, her previous pathology was reviewed which was found to be positive for the following stains (mammaglobin, estrogen receptor, CAM5.2, AE1/AE3, EMA and progesterone receptor with focal syneptophysin immunoreactivity), whereas the current lesion was found to be negative for these markers and stains and thus did not represent metastatic disease. Based on this comparison the lesion was a new separate entity. It showed morphologic and immunohistochemical features of choroid plexus differentiation. The morphology was consistent with ectopic choroid plexus.

Discussion:

Our patient's cystic lesion did resemble imaging characteristics of an arachnoid cyst; minimally enhancing rim, CSF fluid like consistency with no signal restriction to suggest other pathologic entities. However, her history of breast cancer with already known metastatic disease to the bone, and increasing dimensions of the lesion demanded a tissue diagnosis. Even though, the pathologic diagnosis is that of a benign cyst with likely ectopic choroid plexus, the question still remained how did it get there and is it what it is?

Arachnoid cysts are rare and of congenital malformative origin. They typically account for 1% of all intracranial lesions.[1,10] According to Starkman, Brown and Linell, they are thought to form because of abnormal CSF flow that lead to an arachnoid splitting, unless there is a known history of trauma.[3] CSF production by enclosed ectopic choroid plexus tissue is another mechanism of growth, which would suggest a clear cause – cause effect relationship concerning cyst growth. Our patient's lesion is an isolated lesion in the right hemisphere not associated with communicating choroid plexus tissue, with histological and pathological findings of ectopic choroid plexus which likely responsible for the cyst formation. In this case it is unlikely that cyst formation was secondary to abnormal arachnoid splitting.

In previous reports with extensive literature review by Schuhmann and Samii as well as Carter and Singh show only 4 cases of epithelium lined non-communicating cysts incorporating choroid plexus tissue or choroid plexus like tissue were reported and six cases of choroid plexus papilloma that have arisen in a location unassociated with the normal choroid plexus.[1-5,10,12] To our knowledge our case constitutes a 5th reported case of intraparenchymal cystic lesion containing ectopic ChP tissue in an adult. Table 1, obtained from Carter and Singh shows all cases reporting choroid plexus lesions that were not associated with the normal choroid plexus tissue.[12] The one question then and now still remains how did ectopic choroid tissue get there?

In the case of our patient the presentation of ectopic choroid plexus tissue may be explained by the theories proposed by Azzam, Timperly and Greene that choroid plexus cysts might arise from primitive ectopic secretory choroid plexus epithelium that is present within the brain substance but outside the ventricular system (primary ectopia), or from ependymal tissue that became segregated during the developmental stage of the brain (secondary ectopia).[1,4] Since no ependymal tissue was reported by pathologic diagnosis, the possibility of primary ectopia can be considered in our case. Ectopic formation of choroid plexus tissue is not limited to the brain and has been reported in other areas of the body as demonstrated by our review of the literature shown in Table 2.[14-19]

Reviewing the literature, majority of intracranial cysts associated with choroid plexus tissue, were mostly associated with a malignant process based on pathologic evaluation of

surgical specimens. Most diagnosis were consistent with choroid plexus papilloma and one reported case of choroid plexus carcinoma by Carter and Singh. Specific markers and stains were used throughout the literature to differentiate and finalize these findings.

Metastatic carcinomas are characteristically positive when stained with an epithelial non-CK stain and polyclonal antibody against carcinoembryonic antigen, where as our lesion was found to be negative for CK-7. An interesting fact to point out, while these findings point against a possible metastatic disease, it may not guarantee a benign process. Even though, the morphology of our mass was consistent with ectopic choroid plexus with reactive changes, without numerous the well formed numerous papillary structures seen in choroid plexus papilloma are not identified, it cannot be completely excluded.

Similarly, when Carter and Singh evaluated their ectopic choroid plexus carcinoma lesion, it was found to be negative for CK-7 as well.[12] Furthermore, in their review of the literature, they note in a study by Gyure and Morrison, that application of antibodies for CK7 and CK20 to 35 choroid plexus papillomas, majority stained positive for CK7 and CK20, however, six of the tumor were negative for both, including the choroid plexus carcinoma which Carter and Singh reported.[9,12] While our pathological diagnosis suggests benign ectopic tissue, the possibility of choroid plexus papilloma can not be excluded and the need for re-staining for CK20 may be warranted. In addition to CK20, transphyretin (prealbumin) positivity is suggestive of but not specific for choroid plexus differentiation and may be another additional immunostain that could be used in this case or future cases to differentiate a benign lesion from a malignant one.[9,12]

Conclusion:

We reported a case of a supratentorial intraparenchymal cystic lesion incorporating ectopic choroid plexus tissue in a female with known history of breast cancer with metastatic spread to bone. While pathologic evidence definitively excluded this as metastatic spread, and suggested more of a benign process, further evaluation of the specimen with additional immunostains and markers may be of benefit.

To our knowledge this is a 5th reported case of a cystic lesion formation incorporating ectopic choroid plexus tissue. Based on previous reports, the presence of ectopic choroid plexus is the likely cause of cyst formation and the overall increase in mass size, and the ultimate cause of our patient's symptoms. We suggest that recurrence of cystic lesion is likely if incomplete resection of ectopic choroid tissue takes place. Furthermore, we recommended repeat of interval MR imaging to evaluate for lesion recurrence and the need for re-operation and evaluation of tissue.

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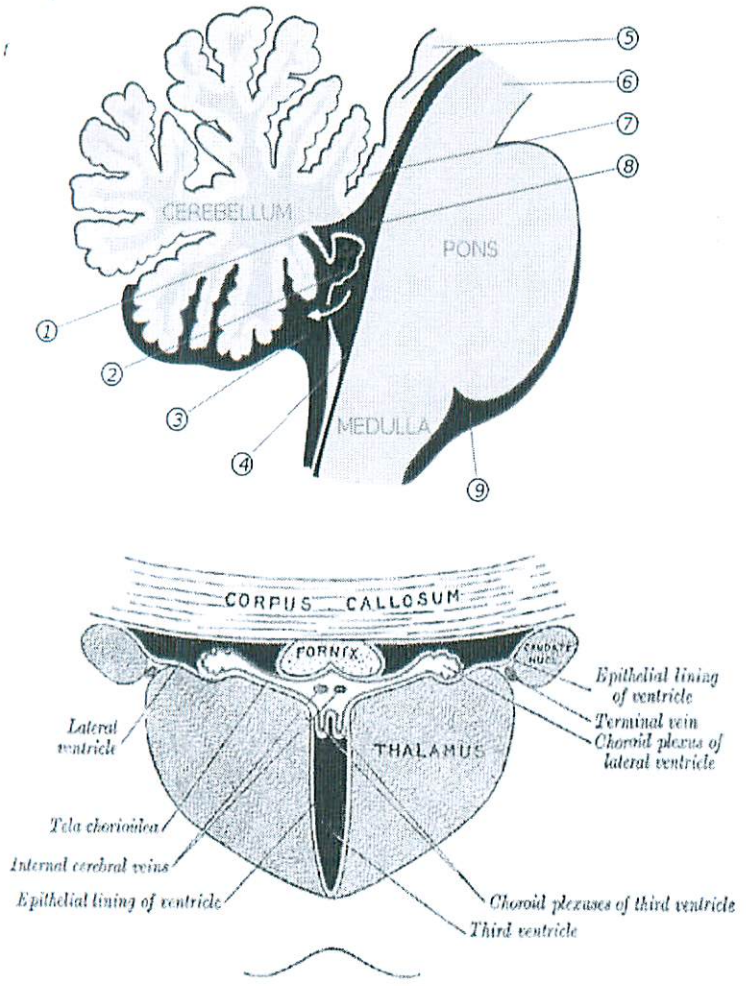

- Posterior medullary velum
- Choroid plexus
- Cisterna cerebellodellaris of subarachnoid cavity
- Central canal
- Corpora quadrigemina
- Cerebral peduncle
- Anterior medullary velum
- Ependymal lining of ventricle
- Cisterna pontis of subarachnoid cavity

Arrow = Flow of cerebrospinal fluid (CSF) through foramen of Magendie

Figure 1. Post embryologic anatomical representation of Ventricular System containing areas of choroid plexus tissue and sites of Cerebrospinal fluid production, direction of flow, filtration and reabsorption.

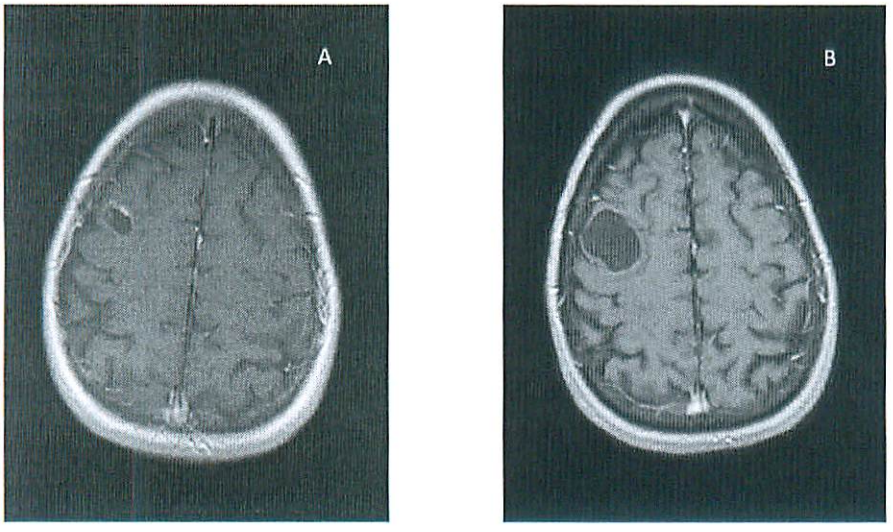

Figure 2. Axial MR images with gadolinium showing a right frontal lobe lesion. **A)** Axial T1 post gadolinium sequence showing intra-axial R. frontal lobe cystic lesion with minimal peripheral enhancement, measuring 1.1 cm x 1.1 cm. No mass affect no midline shift. Evaluated on August of 2003. **B)** Axial T1 post gadolinium sequence demonstrating a hypointense intraaxial right frontal lobe mass with minimal peripheral enhacement, measuring 2.5 cm x 1.8 cm, enlarged in size. Evaluated June, 2010.

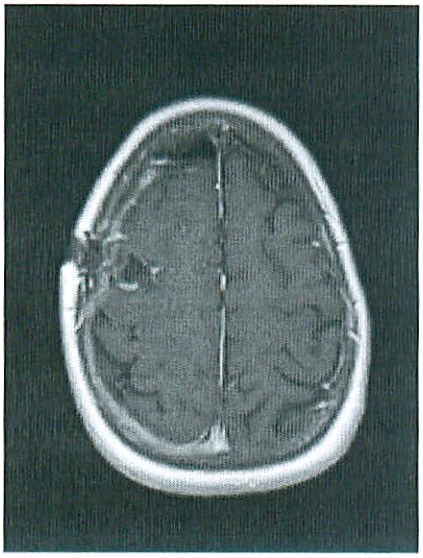

Figure 3. Post surgical MRI T1 axial images post gadolinium infusion. Post surgical changes of right craniotomy and a residual cystic cavity, without evidence of residual wall enhancement. Minimal vasogenic edema surrounding the surgical cavity.

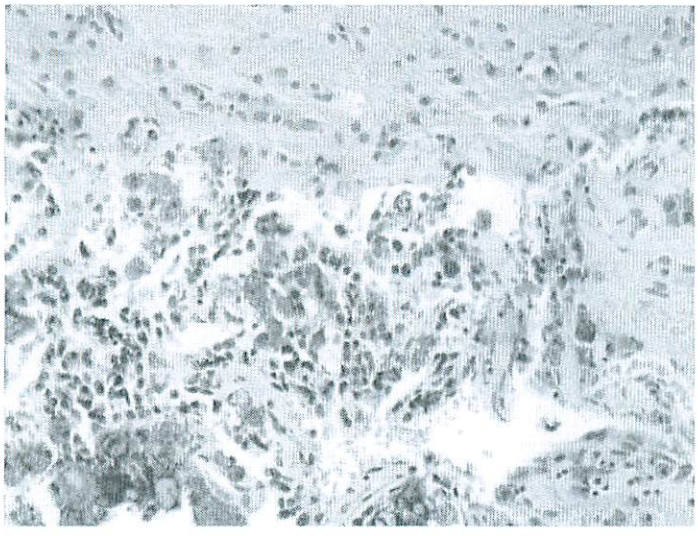

Figure 4. Illustrated is solid portion of the cystic lesion in which fibrovascular papillae lined by uniform bland cuboidal epithelium are identified. Numerous psammomatous calcification are noted. Findings are consistent with choroid plexus tissue. Brain parenchyma (upper and right) shows gliosis and hemosiderin deposition.

Table 1

Literature review of choroid plexus lesions that were unconnected to the normal choroid plexus

Authors & Year	Age, Sex	Type of Lesion	Location of Lesion
Greene, 1951	60 yrs, M	ChPP	rt cerebellar hemisphere
Robinson, 1955	67 yrs, M	ChPP	rt cerebellar hemisphere
Handa & Bucy, 1956	34 yrs, F	choroid plexus cyst	cerebellar vermis
Azzam & Timperley, 1981	32 yrs, F	choroid plexus	yst rt temporal lobe
Inoue, et al., 1987	4 mos, F	choroid plexus cyst	lt cerebral hemisphere
Kimura, et al., 1992	34 yrs, F	ChPP	suprasellar region
Li & Savolaine, 1996	34 yrs, M	ChPP	rt cerebellopontine angle
Nakano, et al., 1997	42 yrs, F	ChPP	posterior 3rd ventricle*

*This ChPP was actually located within the third ventricle but was unassociated with the nearby choroid plexus.

Table 2

Literature review of lesions containing ectopic choroid plexus outside the brain, non- communicating with native choroid plexus tissue

Author and Year	Age	Sex	Type of Lesion	Location
Yapicier & Sawicki (2002)	50 yrs	F	S1-S3 extradural ChPP	Sacral Canal
Tubbs & Georgeson (2003)	2 Wk	M	ChPE, neuroglia, ependymal cells, non – neuroblastic neurons, pigmented retinal epithelium	L. cervical and Face
Dwarakanath & Sharma (2005)	30 yrs	M	ChPE	Intramedullary (C6-T2)
Park et al. (2007)	11 M	F	ChPE	Orbit
Gross et al. (2009)	7 Wk	M	ChPE and cyst	Presacral
Abel et al. (2009)	6 Wk	M	ChPE and other neuroglial tissue	Parapharyngeal
	3 Wk	F	ChP like epithelium and neuroglial tissue	L sphenoid wing, L. infratemporal fossa, L. parapharyngeal space

ChPE = chroid plexus epithelium
ChP = choroid plexus

Treatment of post-traumatic syringomyelia: Silastic wedge "double dorsal fin" draining technique with reconstruction of the subarachnoid space Report of three consecutive cases

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Abstract

Surgical intervention is the preferred treatment option for symptomatic post-traumatic syringomyelia. Most surgical techniques rely on directly draining the syrinx cavity, reconstructing the subarachnoid space or a combination of both to reduce the syrinx size and the patients' symptoms. Studies involving shunting procedures suggest 12-53% of patients improve, 10-56% unchanged and 12-32% regress. Reports of procedures involving only reconstruction of the subarachnoid space show improved or stabilized symptoms in 70-85% of patients and had continued deterioration in 15-30% of patients.

The authors report 3 cases in which the subarachnoid space was reconstructed and the syrinx cavities were drained using a novel technique utilizing two silastic wedges. Historically, shunting procedures have been unsuccessful due to failure of the tubing system used to shunt the syrinx cavity. It is known that tubing is prone to blockage as well as distal or proximal dislodgment. The use of the silastic wedges is thought to limit the adversities associated with using draining tubes to shunt the syrinx cavity. In each case, the clinical history and MRI studies were evaluated to determine the effectiveness of the procedure. All three patients report improved clinical symptoms post-operatively and post-operative MRI studies showed a decrease in the syrinx size along with expansion of the subarachnoid spaces.

Key words: Traumatic syringomyelia, syrinx, spinal cord injury, syringo-subarachnoid shunting, subarachnoid space reconstruction

Introduction:

The precise pathophysiology leading to post-traumatic syringomyelia is still under investigation. Current theories include cystic cavity formations that enlarge from an imbalance between fluid outflow and inflow, as well as cerebral spinal fluid flow alterations in the subarachnoid space caused by arachnoid adhesions, spinal stenosis or spinal cord compression [1,2,3,8,14,17,18,23].

Surgery is the accepted treatment for symptomatic post-traumatic syringomyelia. However surgical approaches differ based on the intended action of the procedure, such as direct drainage of the cavity or reconstruction of the subarachnoid channels or a combination of procedures.

Direct drainage by shunting the syrinx cavity to either the pleural, peritoneal or subarachnoid space has been frequently reported but does not by itself completely correct

the filling mechanism of the syrinx. It essentially ignores the physiological abnormality by only redirecting the flow of fluid outside the syrinx cavity and can be successful as long as continuous drainage is maintained[5,12,13,22,24,26]. Complications including fibrosis with subsequent clogging, shunt migration, and shunt infections have minimized the success of this surgical treatment. Studies have shown variable long term results using shunting as the primary surgical treatment option. Data from long-term studies on shunting procedures suggest 12-53% of patients improve, 10-56% unchanged and 12-32% regress [5,11,19,20,25].

Subarachnoid space reconstruction attempts to recreate a patent subarachnoid plane free of adhesions that provides a channel for normal CSF flow. This is built upon the theory that the inflammatory response following acute injury leads to adhesions in the subarachnoid space and gliosis in the spinal cord. This is followed by a pressure difference above and below the lesion resulting in altered CSF flow [2,5,13,17,21,22,23,25]. Reconstruction often includes duraplasty to expand the subarachnoid space in the area of injury in the attempt to increase CSF flow [5,13,21,22,25,27]. This technique is reported to improve or stabilize symptoms in 70-85% of patients and have continued deterioration in 15-30% of patients in a recent report[25].

In our series, we report the results of three consecutive cases undergoing a combination of laminectomy, subarachnoid reconstruction and syringosubarachnoid shunting with silastic wedges. This series follows an initial case of traumatic cervical syrinx that was shunted with a syringopleural shunt by the senior author. In this case, the shunt failed and eventually led to reoperation and implementation of silastic wedges with lyses of arachnoid adhesions and expansion of the subarachnoid space. This is the current surgical technique used at our institution for treatment of traumatic syringomyelia as reported in the following cases.

Case Reports

Case 1

Presentation and examination: A 35-year-old female with a medical history significant for multiple sclerosis and rheumatoid arthritis developed new onset burning pain wrapping around her thorax just below her mamillary folds. She has a history of lower extremity weakness and paresthesias that correlate with her multiple sclerosis exacerbation. Since the onset of the burning thorax pain, the bilateral lower extremity weakness and numbness had increased in severity. She had also experience new onset bilateral lower extremity thigh pain. On exam, the patient was spastic in all four extremities with movement. Hip flexion, knee extension, plantar flexion and dorsiflexion strength were symmetric but weak (4/5) bilaterally. MRI of the thoracic spine revealed a central syrinx extending from T4-T6 level approximately 3mm in diameter and 50mm in length.

Operative: T5-T7 laminectomy with silastic wedge syringosubarachnoid shunting and reconstruction of the subarachnoid space was performed. Focal arachnoid scarring was

present and the spinal cord pulsations were attenuated. Following lysis of adhesions and reconstruction of the subarachnoid space, the thoracic spinal cord was noted to have increased pulsations.

Post-Operative course: In the immediate post-operative period, the patient noticed a significant decrease in the severity of her thorax pain. The lower extremity pain had resolved and her lower extremity strength had improved to the point she could walk again with a cane. At 8 months post-op the burning pain had completely resolved and the weakness and paresthesias returned to her baseline that she experiences with MS exacerbations. Overall, the patient was pleased with the results of her surgery. MRI showed a decrease in the size of the syrinx cavity. At 8 months post-op it measured 2.0mm in diameter and extended 40mm in length.

Case 2

Presentation and examination: A 38-year-old female with progressively worsening intractable neck pain and headaches, who had previously been involved in a motor vehicle accident, underwent a C4-C7 anterior cervical discectomy and fusion for cord compression and myelopathy. At that time it was noted that a small syrinx was present in the lower cervical cord. Since her original operation she has been taking multiple narcotic medications including 80my of Oxycontin daily to control her neck pain and headaches. On examination the patient had no focal neurologic deficits however she was hyper-reflexive in all extremities. MRI of the cervical spine revealed a central syrinx extending from C5-C6 approximately 3mm in diameter and 20mm in length.

Operative: C4-C7 posterior cervical decompression and fusion with silastic wedge syringosubarachnoid shunting and reconstruction of the subarachnoid space was performed. Focal arachnoid scarring was present and damped spinal cord pulsations were noted. Following reconstruction the cervical spinal cord was noted to have increased pulsations.

Post-Operative course: In the immediate post-operative period the patient's headaches and neck pain improved and her narcotic use decreased. The post-operative period was complicated by an altercation in which the patient experienced new onset neck pain, right arm and leg pain. MRI showed a decrease in the size of the syrinx cavity. At 4 months post-op it measured 2mm in diameter and 20mm in length.

Case 3

Presentation and examination: A 50-year-old female with a history of previous MVA and T10-T12 laminectomy and fusion in 2004 for cord compression due to a herniated disc. She presents 5 years later with new symptoms of progressively worsening right arm pain, burning pain in the back of the shoulders and neck along with intractable headaches. On exam she was found to be hyper-reflexive bilaterally in the upper and lower extremities. Positive Hoffman's sign bilaterally. No focal strength deficits. MRI of the

cervical and thoracic spine revealed herniated discs with cord compression at C5-C7 with a central syrinx extending from C6-T1 measuring 45mm in diameter and 57mm in length. A C5-C7 anterior cervical discectomy and fusion was initially performed to decompress the spinal cord. The radicular component in her right arm resolved however, the headaches and burning pain in the shoulders and neck were still present. The patient was taking vicoden and lyrica for pain control.

Operative: C5-T1 posterior cervical decompression and fusion with silastic wedge syringosubarachnoid shunting and reconstruction of the subarachnoid space was performed. Focal arachnoid scarring was present and spinal cord pulsations were attenuated. Following reconstruction the cervical spinal cord was noted to have increased pulsations.

Post-Operative course: In the immediate post-operative period the patient experienced a resolution of the majority of her symptoms. She was able to wean off of all narcotic medications and was pleased with the results of the surgery. MRI showed a decrease in the size of the syrinx cavity. At 5 months post-op it measured 22mm in diameter and 35mm in length.

Results:

In all three cases the patients reported improvement in their clinical symptoms and there was no report of neurologic deficit from the procedure or progression of the syrinx at 6 months follow up. One case was complicated by an injury sustained during the post-operative period, which resulted in a relapse of narcotic use.

The radiographic evidence of decreasing size of the syrinx cavity and expansion of the subarachnoid space can be seen in figures 1-3.

Figure 1: Left preoperative. Right ten months post op.

Figure 2: Left preoperative. Right four months post-operative.

Figure 3: Left preoperative. Right six months post-operative.

Discussion:

We believe that optimal surgical results can be obtained when both the anatomical and physiological abnormalities associated with syrinx formation are corrected.

In each case, reconstruction of the subarachnoid space with lysis of adhesions was undertaken with the aid of an operating microscope. Tedious care was taken to keep the subarachnoid space free from proteinaceous substances that may lead to future scarring. After a midline myelotomy was performed at the thinnest posterior-inferior aspect of the cavity, two silastic wedges were placed side-by-side and secured to the pia with non-absorbable suture as seen in figure 4. The purpose of the wedges is to maintain patency between the syrinx cavity and the reconstructed subarachnoid space by preventing

healing of the myelotomy edges and to maintain an artificial conduit between the syrinx cavity and the subarachnoid space.

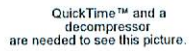

Figure 4. Intraoperative photo of the two silastic wedges sutured in place.

The silastic wedges allow for the passage of cerebral spinal fluid through capillary action under very small pressures without collapse. The benefit to this method is conferred as the syrinx cavity is drained and pressures within the cavity become less. In a traditional shunt procedure with tubing, a higher pressure must be maintained to achieve continued flow. As fluid is drained the pressure decreases and the cavity collapses around the perforations in the shunt tubing [5,13,21,22,24,27]. Flow through the tube becomes stagnant and the susceptibility to clogging, which is a well-recognized complication with shunting procedures, increases [13,21,22]. With the silastic wedges, a potential space is created between the two surfaces allowing a constant channel for fluid to pass under changes in pressure and cavity size. The low profile of the silastic wedges within the subarachnoid space may also help reduce subarachnoid adhesions that have previously been reported with using tubing to shunt the syrinx cavity [5,13,21,22,24,27]. When securing the tubing into place, the round nature makes it difficult to suture to the pia. The flexible silastic wedge allows for a simple stitch that can be placed under direct visualization as to avoid damaging any surface vessels. The trigone shape of the wedge allows for direct visualization and safe placement into the syrinx cavity without blind advancement. Additionally, we also increase the compliance of the subarachnoid space by lysing adhesions and duraplasty to reduce any pressure gradients that may form and alter the fluid dynamics between the syrinx cavity and subarachnoid space [2,5,12,13,14,21,22,25,28].

Our surgical approach used to treat symptomatic traumatic syringomyelia has evolved along side our interoperative experience, surgical outcomes and evaluation of the literature. The initial complication we incurred with shunting the syrinx to an pleural space led to the adaptation of our shunting technique. The inconsistent results that are reported with shunt tubing leads us to question whether the fluid is truly draining through the shunt tubing. Because a consistently high pressure would be required to maintain flow, we believe that the CSF is egressing around the shunt tubing into the subarachnoid space rather than draining through the tubing. We have found that the silastic wedges provide a similar conduit requiring less pressure to drain fluid from the cavity. The shape and pliability of the wedges lend to a one step safe insertion that does not require blind

advancement or twisting into the syrinx cavity. It also provides greater flexibility when securing to the pia.

Conclusion:

The short-term results of the silastic wedge syringosubarachnoid shunt with subarachnoid space reconstruction have shown promise in relieving clinical symptoms and reducing syrinx size. However, long-term follow up is needed to further evaluate the ultimate success of this procedure.

P1-P1 Stent Construct for Coil Embolization of Basilar Apex Aneurysms: Technical Note

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Introduction

Embolization of basilar apex aneurysms (BAA) with successful long-term outcomes is a challenging endeavor with regard to its often wide-necked eccentric relationship with the PCA origins and high rate of recurrence. We present three cases from our series of basilar apex aneurysm residuals that were retreated using stent assisted coil embolization. Stent placement was achieved by delivering it from P1-P1 segments of the posterior cerebral arteries via microcatheterization across a patent posterior communicating artery

Endovascular Technique with Illustrative Cases

Three basilar apex aneurysms previously treated with coil embolization showing recurrence were retreated using stent assisted coil embolization with stent placement from the P1-P1 segments of bilateral PCAs. The stent placement and coil embolization were done as two stage procedures allowing for adequate endothelialization of the stent. Both Neuroform and Enterprise stent systems were used during separate cases along with their respective delivery microcatheters.

Procedures were performed under general endotracheal anesthesia. All patients were given a pre-operative aspirin (325mg) and Plavix (75mg) anti-platelet regimen in anticipation of stent placement. A baseline ACT (activated clotting time) was drawn at the start of the procedure and a 75unit/kg heparin bolus was given to achieve a working ACT longer than 250 seconds. Subsequent ACTs were performed throughout the procedure to verify adequate heparinization and re-dose as needed. Post procedure all patients were kept on a low dose

(400units/hr) heparin drip overnight to prevent microembolic complications associated with the fresh stent. All patients continued a dual anti-platelet regimen 3 months post procedure and were continued on aspirin only thereafter.

After verifying no prior groin complications from prior angiography access, bilateral femoral artery access was obtained. A 6F Envoy MPD guide catheter is placed into the internal carotid artery with the larger patent posterior communicating artery (PCOMa) under roadmap guidance, and a Simmons 2 5F diagnostic catheter is passed through the other groin sheath to select the vertebral artery for posterior circulation injection. Simultaneous injection of the guiding catheter and diagnostic catheter provides a working roadmap to navigate the synchro 2 microguidewire and respective delivery microcatheter (Prowler Select Plus or Renegade Hi Flo) from the anterior circulation across the PCOMa on AP and Lateral imaging. Next the microwire is advanced into the posterior cerebral artery on the ipsilateral side and crossed along the base of the aneurysm across the basilar apex into the left posterior cerebral artery. We advance to approximately the P2-P3 junction, the microcatheter is advanced over the wire into the mid P2 segment of the contralateral PCA. Following this maneuver we remove the wire and load the stent along the same trajectory. We unsheathe the stent from P1-P2 junctions bilaterally thereby bridging the neck of the aneurysm and protecting the basilar artery and bilateral P1 segments.

3 months post stent placement after adequate stent endothelialization the second stage of coil embolization is undertaken. This time a 6F guiding catheter is placed into the working vertebral artery and after appropriate anticoagulation the basilar apex aneurysm is accessed utilizing a transcend 14 microwire and SL-10 microcatheter for coil embolization, during the embolization stage on lateral imaging the outline of the stent profile provides an additional advantage in that it outlines the anatomical endpoint for termination of coiling and allows the surgeon to achieve maximal packing density without compromising parent vessels. This down the barrel view is helpful as often times the tilt and overlap of existing coils in the aneurysm obscures the true neck of the aneurysm on AP projection.

All three patients who received a P1-P1 stent and subsequent coil embolization of their basilar apex aneurysms demonstrated gross total radiographic occlusion of their aneurysms from the intracranial circulation. Additionally, the normal intracranial vasculature was preserved with excellent flow distal to the stent markers, with no evidence of in-stent stenosis or migration.

CASE # 1: JK

63 y/o female presented with a subarachnoid hemorrhage. She underwent coil embolization of a 14mm x 15mm basilar apex aneurysm on 4/17/07, initially with balloon protection of both posterior cerebral arteries. The aneurysm was successfully coiled with gross occlusion. 3 month follow-up angiography showed a large recurrence. The recurrence was treated with stent assisted coil

embolization utilizing a P1-P1 deployment of an Enterprise stent. 3-month follow up angiography shows gross total occlusion of the aneurysm.

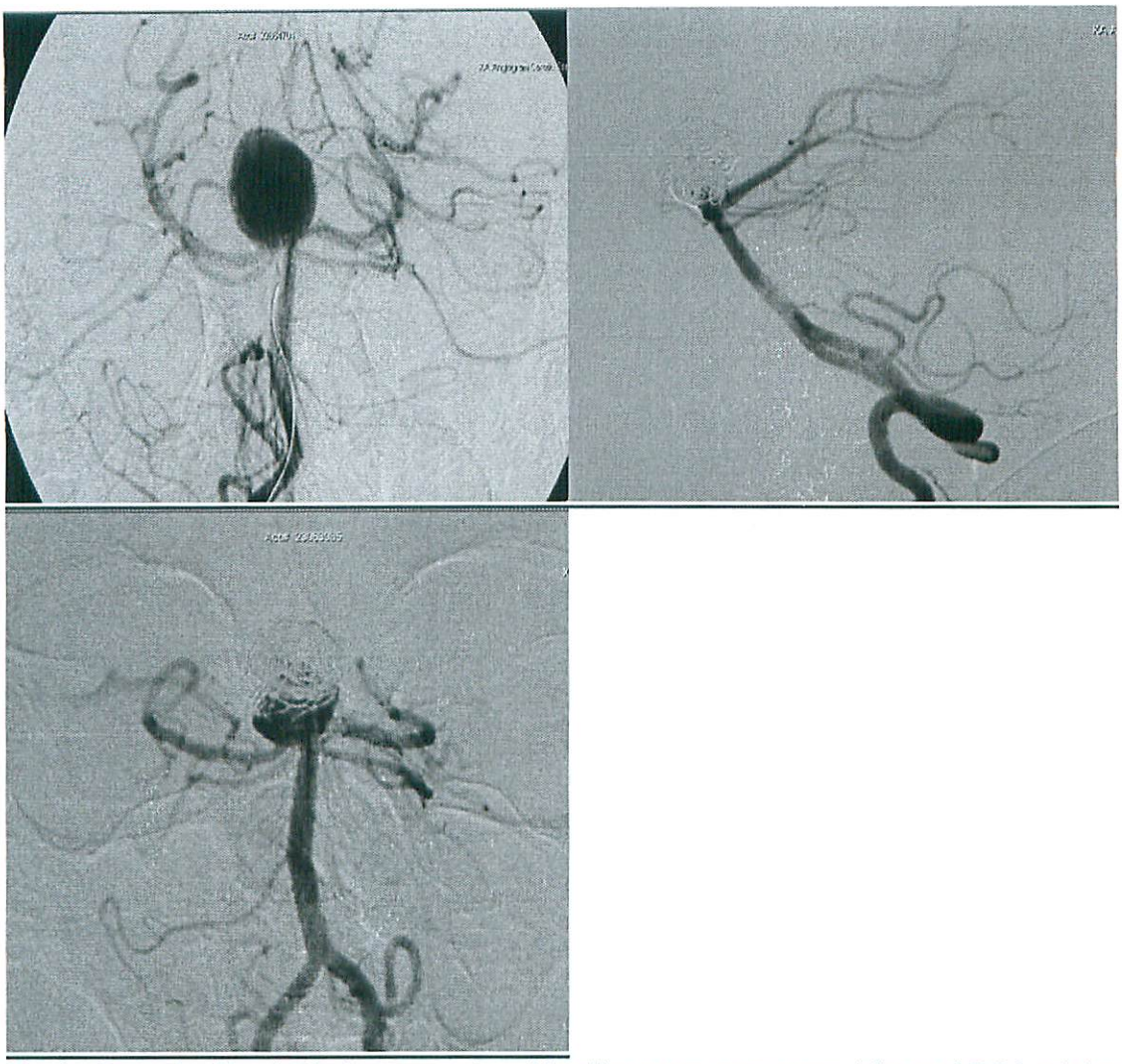

Fig 1. Case 1. *Top Left:* Large ruptured basilar apex aneurysm, bilateral PCA origins are involving the neck of the aneurysm. *Top Right:* Lateral view showing gross total occlusion after initial coil embolization of basilar apex aneurysm with balloon assist. *Bottom Left*: AP projection showing coil compaction and recurrence of previously coiled aneurysm overlying the origins of bilateral PCAs as well as the right SCA

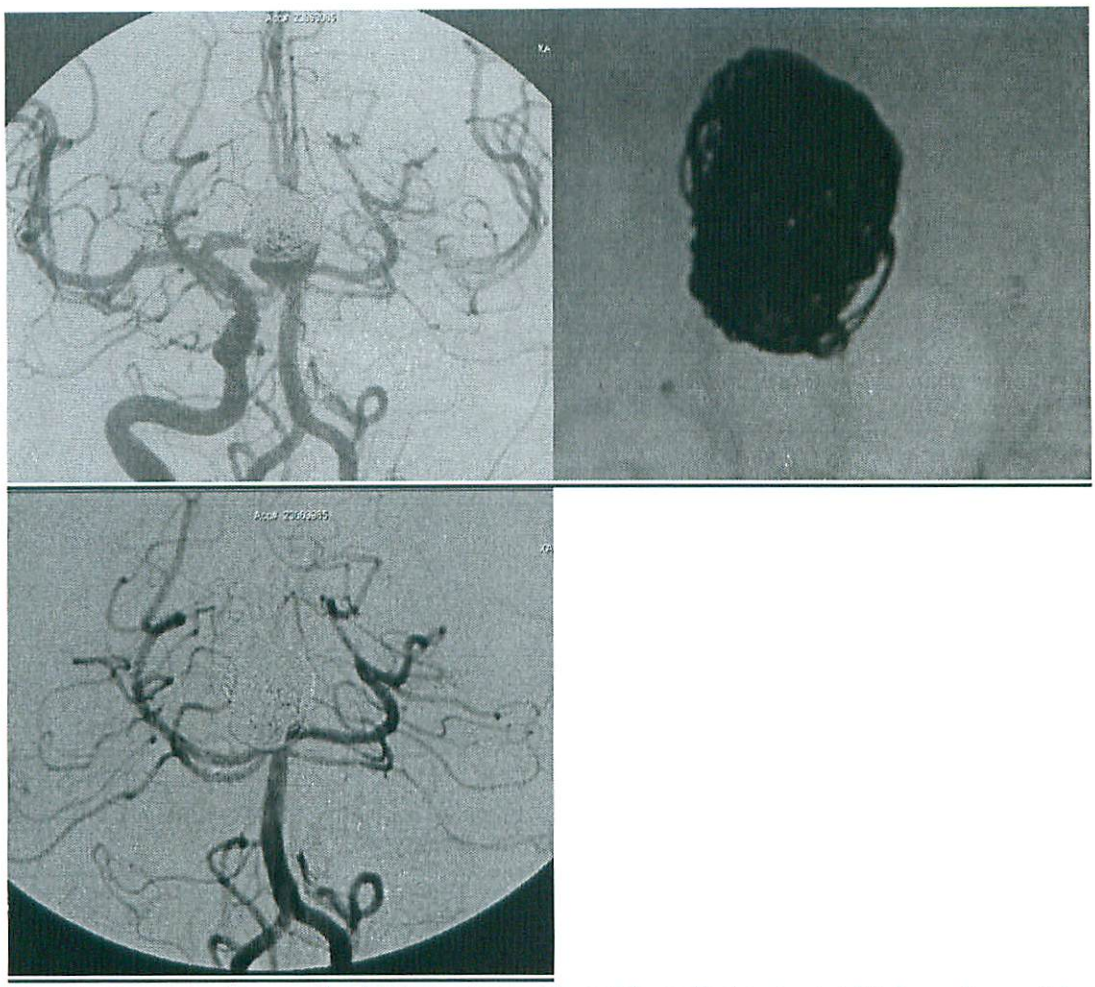

Fig. 2. Case 1. *Top Left*: AP injection with guide catheter in right internal carotid artery and diagnostic catheter in left vertebral artery for planning of stent placement. *Top Right*: AP image showing stent-coil construct. The stent is bridging the aneurysm neck and protecting bilateral posterior cerebral arteries. *Bottom Left*: AP image at 3 month follow-up angiography after P1-P1 stent assisted coil embolization of basilar apex aneurysm recurrence demonstrating exclusion of the aneurysm from the normal intracranial circulation.

CASE # 2: DS

63 y/o female with a ruptured large basilar apex aneurysm previously treated at an outside hospital with a recurrence after multiple retreatments. The patient came to us from Florida with an MRA showing the latest recurrence with residual at the neck and flow within the coil mass. This was treated with a Neuroform 3 stent across P1-P1 junction followed by coil embolization in staged procedures. 6 month follow-up angiography revealed gross total exclusion of the aneurysm with preservation of the normal intracranial circulation

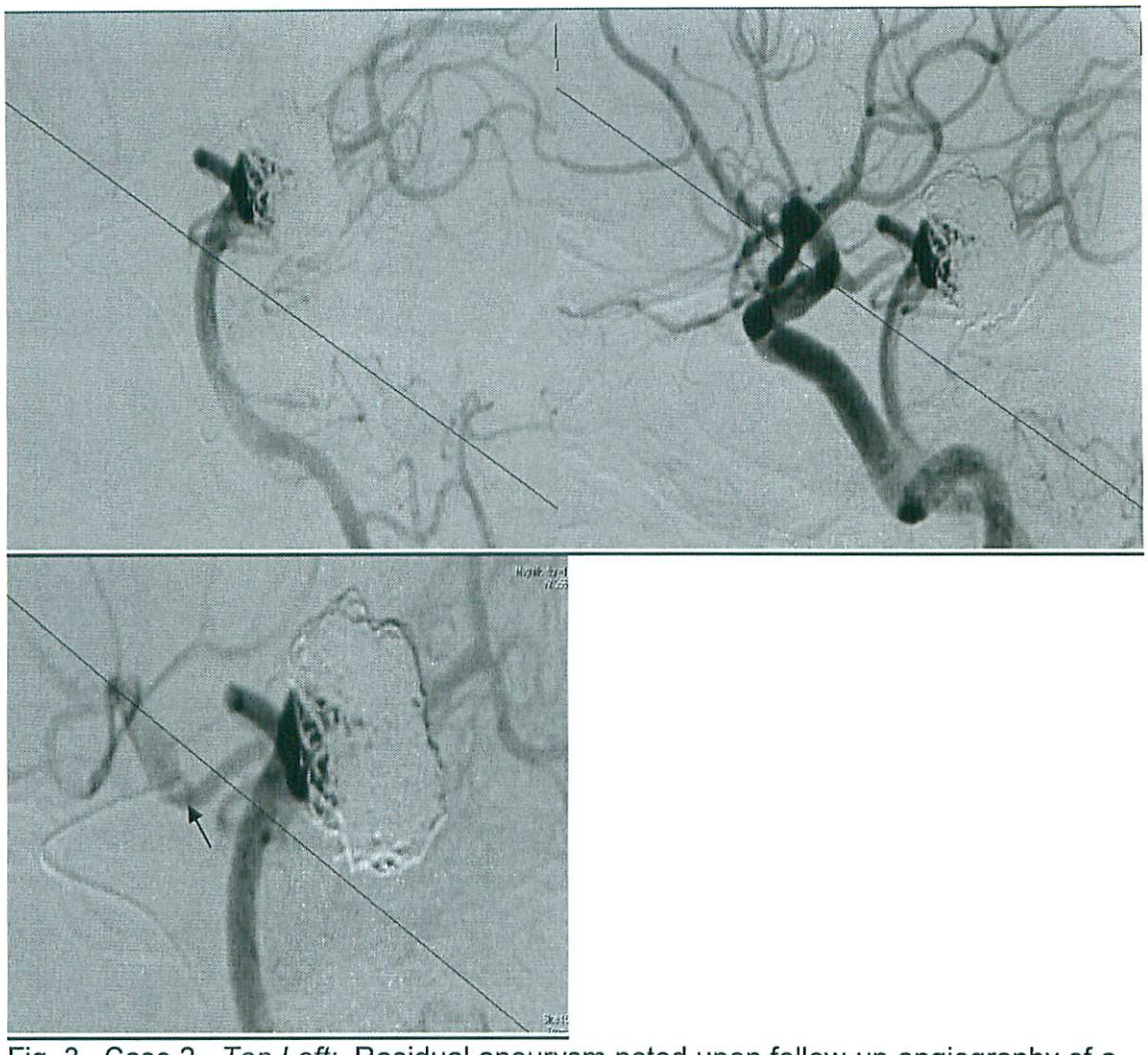

Fig. 3. Case 2. *Top Left:* Residual aneurysm noted upon follow-up angiography of a previously treated basilar apex aneurysm. *Top Right:* Lateral Projection showing simultaneous injection via right carotid and vertebral arteries prior to microcatheterization of posterior communicating artery. *Bottom Left:* Lateral projection after vertebral artery injection note tip of microcatheter (black arrow) positioned at ostium of the posterior communicating artery prior to advancement of micro wire.

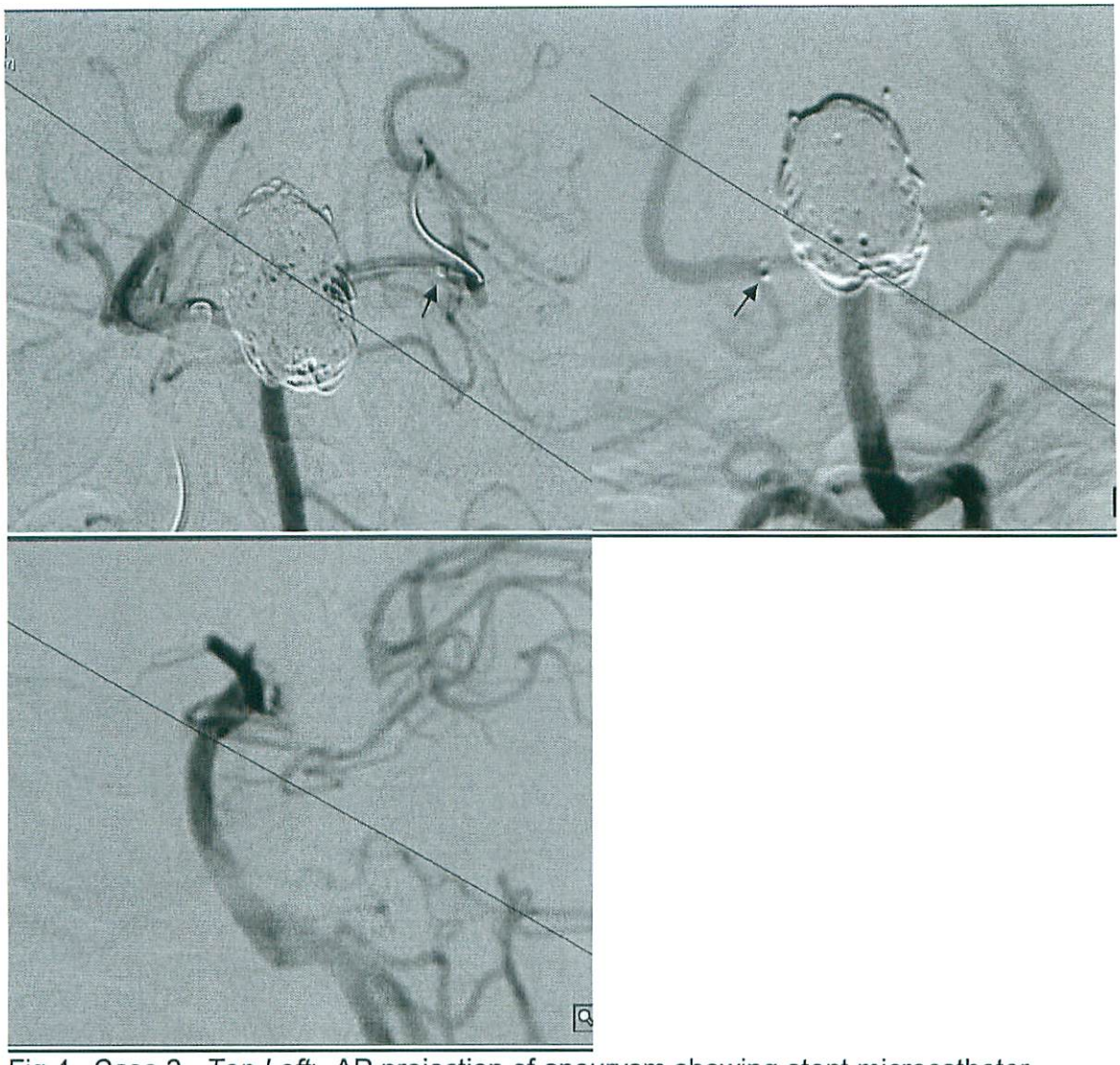

Fig 4. Case 2. *Top Left*: AP projection of aneurysm showing stent microcatheter traversing anterior to posterior circulation. Tip of microcatheter (black arrow) is in the proximal left P2 segment with microwire extending into the distal P3 segment. *Top Right*: AP view of basilar apex aneurysm after stent deployment showing coil mass and stent construct, note proximal stent markers (black arrow) seated at junction of right PCA and PCOM arteries. Also note the classic "T-shape" appearance of PCA takeoffs with large posterior communicating artery, an anatomic setup optimal for the P1-P1 stent. *Bottom Left*: Lateral view of vertebral injection post stent assisted coiling showing gross total occlusion of basilar apex aneurysm

CASE # 3: MS

49 year old male who had coil embolization of a ruptured basilar apex aneurysm 11/15/2001 with gross total occlusion at the initial treatment, was noted to have a 5mm Type II recurrence on 18 month follow up imaging. The recurrence involved the origins of both PCAs. He underwent P1-P1 Enterprise stent placement followed 3months later by coil embolization of the residual aneurysm. 6 month follow up angiography showed a stable appearance to the aneurysm

that was retreated with stent assist. Patient was doing clinically well and had returned to work with no gross neurological deficits.

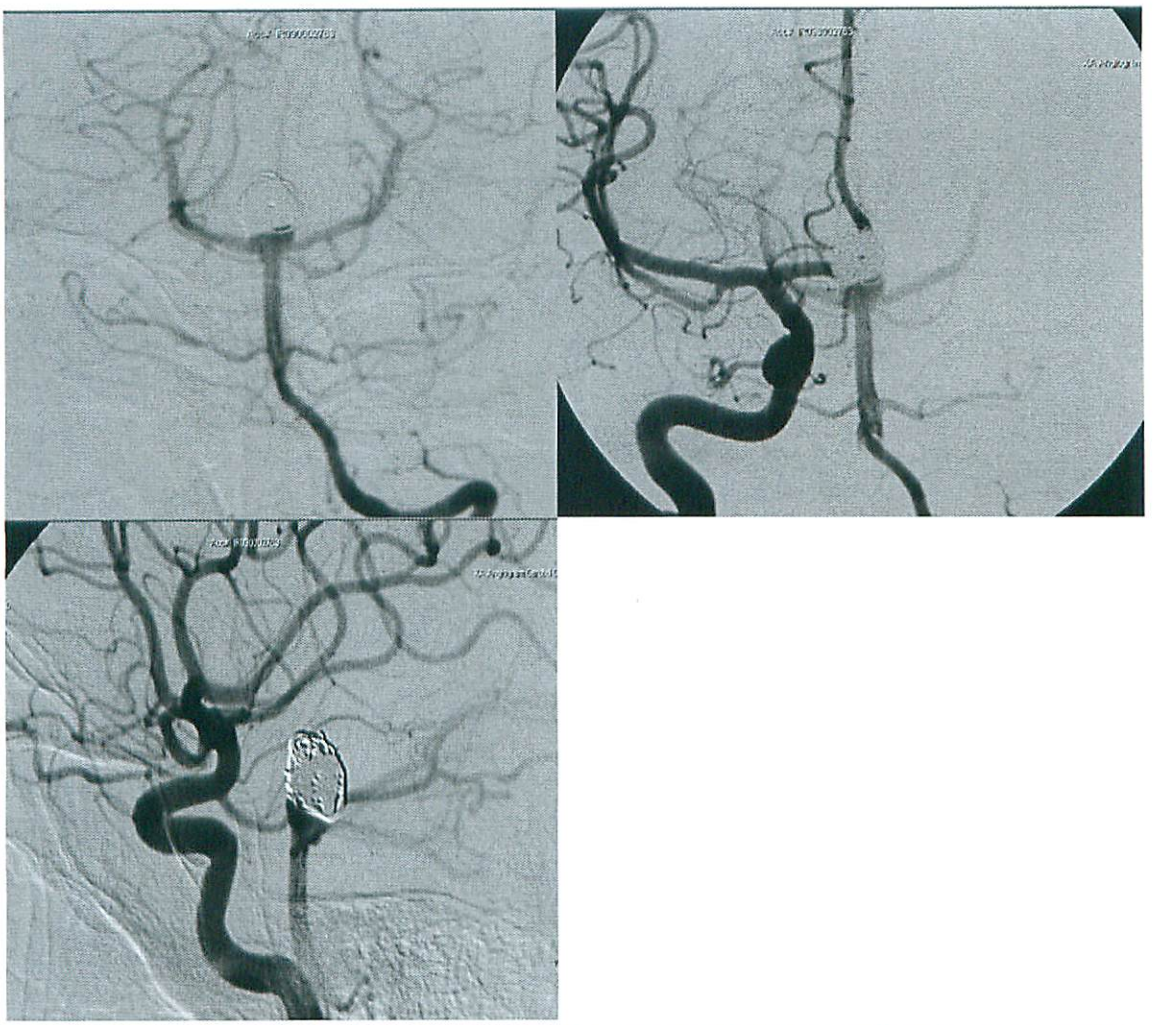

Fig 5. Case 3. *Top Left:* Type II residual of previously treated basilar apex aneurysm with a wide neck involving PCA origins. *Top Right:* AP projection after injection of right carotid and left vertebral arteries showing planned roadmap for delivery of stent via right posterior communicating artery. *Bottom Left:* Lateral Projection showing planned roadmap for navigation of a microcatheter for stent deployment via a large patent posterior communicating artery

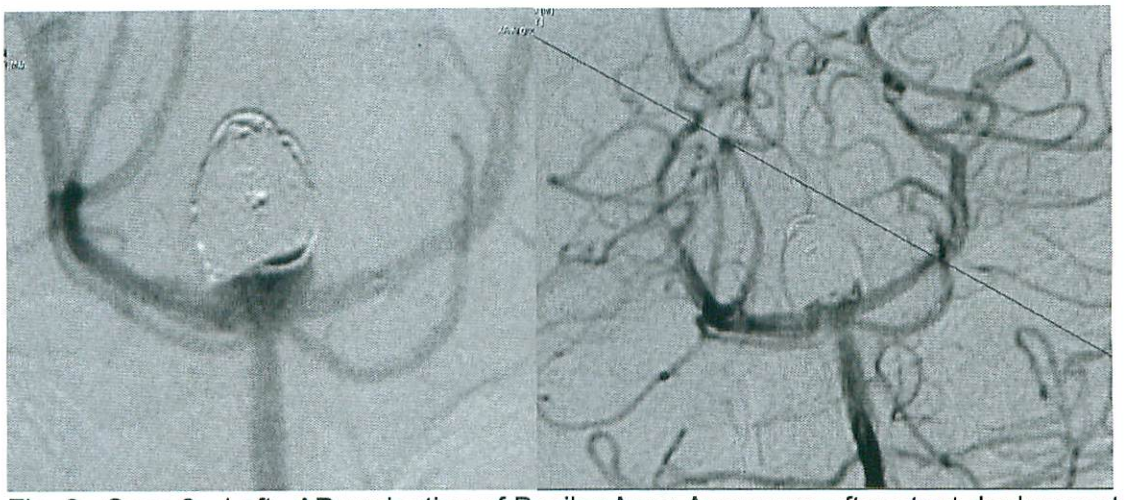

Fig. 6. Case 3. *Left:* AP projection of Basilar Apex Aneurysm after stent deployment showing stent markers in bilateral posterior cerebral arteries. *Right:* 6 month follow-up angiogram: Basilar apex aneurysm after retreatment with stent assisted coil embolization

Discussion

Anatomic and technical constraints aside, the utility of this stent construct should not be underestimated and long-term comparisons of BAA recurrences and coil compaction comparing this construct relative to others is needed to show what is thought to be improved packing volumes and lower recanalization rates with the P1-P1 stent. This is not a novel description of stent assisted coil embolization and a review of literature has been noted for so called "horizontal stents" or "circle of Willis stents", however a sample of our series of P1-P1 stents is shown to add to the pre-existing literature and to share our technique and encountered pitfalls. In addition, as more high volume endovascular centers engage each other and share data and experience, the overall outcomes and long term results can be given a validated quantitative value as opposed to limited low sample size case series.

Benefit of P1-P1 Construct

The added benefits of this construct are as follows:

Cost reduction - by using one stent to protect both PCA origins versus the use of the "Y" stent configuration that incurs extra cost in the order of thousands of dollars.

Optimal visualization - stent mesh outline gives better visualization of true aneurysm neck upon coil embolization. The lateral projection provides a "down the barrel" view across the basilar apex visualizing both PCA origins, although this does become more difficult with the more obtuse angled and "bat-winged" PCA configurations. As noted from prior reports the optimal anatomic setup for

this stent construct would be a large PCOMa and a "T-shaped" configuration of the PCAs relative to the basilar trunk.

Ease of aneurysm access - navigating across a single stent is much simpler and safer, regardless of whether a closed cell or open cell system is used as only one "layer" of stent mesh needs to be crossed by the microcatheter to access the aneurysm. The neurovascular stents were designed to allow access for a single 3F maximum outer diameter microcatheter through its interstices. In the Y stent configurations the cris-crossing of the two stent meshes reduces the maximum available space to negotiate a microcatheter through, in addition, the curved trajectory of deployment of the Y-stent also can pinch close the cells on the concavity of the bend, particularly in the open cell design.

Straight deployment of the stent - this technique avoids deployment of the stent in a curved trajectory thereby limiting the occurrence of stent strut prolapse and unequal stent delivery that is often associated with deployment of open celled stents in curved vasculature (Benndorf). The unavoidable discrepancy in size of the basilar trunk and P1 segment converts to unequal opening and wall opposition of the stent that can lead to higher rates of in-stent stenosis and stent migration. A straight stent in this area gives a more natural contour to the true aneurysm neck and protects the basilar trunk, bilateral PCAs and any other branching arteries such as the SCA as shown in Case 1 which may be compromised by the neck of the aneurysm.

Higher Packing volume - the above mentioned benefits of the stent construct adds to the security needed to aggressively and safely pack the aneurysm to gross total radiographic occlusion. The general consensus from our experience and others' from small case series that have been reported is that this construct will lead to higher coil packing volumes and more durable long-term results, but cooperative data and outcomes need to be compiled to evolve from anecdotal to scientific statements.

Failures of "Y stent" construct

Many surgeons have incorporated the "Y" stent reconstruction of the basilar apex to aid in coil embolization of basilar apex aneurysms that need protection of bilateral PCA origins. This technique although shown to be efficacious does have its technical challenges and associated morbidity. To start with, the obvious expense of 2 stents vs. 1 stent for reconstruction of the aneurysm neck has its cost drawbacks. Technically, the deployment of 2 stents requires extra steps involving deploying the first PCA-Basilar stent then re-accessing the contralateral PCA through the first stent, accurately deploying the second stent to match the proximal length of the first stent, then accessing the aneurysm via the Y construct whose cells have been deformed by the natural curves of the vasculature and the overlapping stent laces. There are also known complications associated with stent placement in this particular configuration

including stent migration due to the unavoidable discrepancy in size between the basilar trunk and the P1 segments creating the "watermelon seeding" effect, particularly with closed cell stents (Kelly). The changes in laminar flow within the basilar trunk associated with the unequal crossover may predispose to in-stent thrombosis and in- stent stenosis. As described above, re-accessing the aneurysm through the deformed interstices of the two stents is also more challenging relative to crossing a single stent.

Technical Difficulty

As with all surgical procedures the ability to perform said technical notes and reproduce them with the same safety and efficacy is of the utmost importance. There are inherent technical hurdles that will be encountered during this procedure and the risks associated with these should always be in the forefront of the decision making process.

The necessity for double groin puncture is the first step that can add to the cumulative morbidity from local complications. Anterior circulation and posterior circulation access is needed for this procedure. The stent stage requires a diagnostic catheter for the posterior circulation injection and a guide catheter for the anterior circulation access. Local pathology in those regions such as stenosis, tortuousity of vessels from the arch on up, occlusion, can all be limiting factors.

The second hurdle is navigating across the posterior communicating artery from anterior to posterior circulations. The inherent size of the PCOMa can be a limiting factor needing to abort the procedure due to vessel/microcatheter mismatch. As for navigation across any intracranial vessel, the PCOMa must be at least 2mm in size to allow the microcatheters to pass with ease. An issue that has been encountered is the small but non-negligible discrepancy in size between the Renegade Hi Flo and the Prowler Select Plus microcatheters. The Enterprise stent is delivered through the Prowler Select Plus microcatheter which has a 2.3F distal outer diameter, compared to the 2.8F outer diameter of the Renegade Hi Flo microcatheter through which the Neuroform stent is delivered; however preference of open over closed cell stent systems may require the practitioner to use a larger delivery catheter.

Prior to the latest engineering change in the delivery style of the Neuroform stent the most challenging step of delivering this stent was the need to perform a microcatheter exchange of the microcatheter used to navigate across the circulations to the preloaded Renegade microcatheter.

Other navigation issues that come up are the ability or inability to maneuver across the PCOMa, through the ipsilateral P1, across the neck of the aneurysm (basilar apex), select the contralateral P1 and negotiate the microwire into the contralateral P2-3 segments. Subsequently the trackability of the entire delivery

system will limit the ability to deliver the preloaded stent to the intended deployment zone.

With regards to stent deployment one must keep in mind a few things. There is a theoretical increased risk of "watermelon seeding" and stent migration of the closed cell system (Enterprise) and this risk is compounded by the fact that the deployed stent will likely be of a smaller length to keep the ends deployed within the P1 segments symmetric. Therefore accurate distal deployment and engagement of the distal P1 and proximal P1 is needed as the average P1 length is approximately 6mm.

It is important to note that the proximal end of the stent should engage just short of the P1-PCOMa junction as the delivery catheter is limited by the course taken across the PCOMa. We try to engage the contralateral P1 segment at the same distance for two reasons; the first being that optimal stent placement will be just short of the PCOMa-P1 junction so as to reduce the surface area of vessel covered by metal in the case of acute thrombosis or long-term intimal hyperplasia, thereby reducing the potential for complete PCA occlusions without flow across the circle of Willis; the second is to limit the possibility of unequal force distribution on either end of the stent and potential catastrophic stent migration. There may be an asymmetry in the diameters of the P1 segments. This in addition to asymmetric stent deployment and a short potentially closed cell stent could lead to a series of unfortunate events.

Conclusion

P1-P1 stent placement for coil embolization of complex basilar apex aneurysms is a safe and effective alternative to other stent constructs such as the P1-Basilar or Y-stent configuration. Proper patient selection is needed based on size of the residual aneurysm, morphology of the PCA takeoff relative to the neck of the aneurysm, adequate sized PCOM to navigate a microcatheter across, and using it for unruptured or for retreatment of previously ruptured aneurysms.

This stent construct allows for better protection of the origins of vessels arising from the basilar apex, provides a stable scaffold for higher coil packing volumes, is cost-effective avoiding the need for multiple stents for reconstruction of the basilar apex, provides a "down the barrel" visualization of the neck of the aneurysm and PCA origins during coiling which enhances the surgeons ability to protect the PCA origins while achieving maximum aneurysmal packing. Staging the stent delivery and coiling in two procedures to allow adequate stent endothelialization will aid in the success of this procedure. Long-term cooperative outcomes on these and other "novel" techniques should be sought out to give validity to their use and make them a standard of care not just a novelty.

Disclosure/Disclaimer

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