CV Section News

Chairman's Message

Editor: Carlos David, MD
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It is a great honor to have the opportunity to take the helm of the Cerebrovascular Section following the lead of Sander Connolly who has steered us with great poise over the last year. I fully anticipate smooth sailing in many aspects of the Section's path, given the energetic and highly capable group of individuals serving on our executive committee, but we also have to occasionally batten down the hatches to prepare for on-coming storms.

Publication of recent trials such as COSS (Carotid Occlusion Surgery Study), and SAMMPRIS (Stenting vs Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis), are sure to have an impact on the management of ischemic disease, and yet must be taken in context of the confined parameters of randomized study populations. The Section must continue to take an active role in ensuring that results of such studies are not inappropriately over-generalized. An editorial on behalf of the CV Section is now on-line and soon to be published In Neurosurgery regarding the COSS trial, aimed at providing an in-depth critique of the study and its implications for those refractory hemodynamically compromised patients falling outside the study population. The NEJM publication of the SAMMPRIS study heralded the unexpected consequence of a public advocacy group call for removal of the Wingspan stent Humanitarian Device Exemption FDA approval. Such an action would leave symptomatic intracranial stenosis patients failing best medical therapy, and their practitioners, with no options. Andrew Ringer led a rapid response on behalf of the CV Section outlining our opposition to withdrawing FDA approval, and along with Peter Rasmussen represented our position at the FDA Neurological Devices Panel. We can anticipate that the early halting, in May, of the Interventional Management of Stroke III Trial (IMS-III, examining IV tPA alone vs intra-arterial interventions in acute stroke) for reasons of futility may be mis-construed as a serious blow to a promising area of stroke intervention; rather, the limitations of the study design (which did not specifically select patients with large vessel occlusion, and utilized a wide array of devices) must be fully examined once trial results are published, and placed into appropriate context in order to inform the next steps in investigation. The Section, led by Kevin Cockroft, also recently joined with our partner organization, the Society of Neurointerventional Surgery (SNIS), to draw attention to the shortcomings of the ongoing ARUBA study (A Randomized Trial of Unruptured Arteriovenous Malformations) in an editorial piece currently published online in Stroke. For the sake of advancing care for our patients, ultimately we need to remain engaged not just as onlookers, analyzing study design and results, but in active participation and leadership of future trials. The Section is uniquely positioned to harness the expertise of endovascular, open vascular, and those with combined skills, to direct future scientific trials that will guide us going forward.

In line with our goal to pursue a forum for robust scientific exchange and promote the cutting edge in both the endovascular arena and the surgical realms, we can anticipate what is sure to be spectacular Annual Meeting this year. We have joined forces with SNIS to organize a joint meeting which is to be run in Hawaii on February 3rd – 5th, immediately preceding the ISC meeting (Feb 6th -9th), with program committee chaired by Ketan Bulsara (CV Section) and Don Heck (SNIS). The meeting will feature sessions focused both on ischemic and hemorrhagic disease, and we are also looking forward to cross-pollination with colleagues from Japan and the Pacific Rim attending their Joint Neurosurgical Convention in Hawaii on January 29th to February 3rd. I hope to see our membership out in force to take advantage of the incredible meeting content and venue.

Sepideh Amin-Hanjani, MD FAANS FACS FAHA

A TRIBUTE TO C. MILLER FISHER

by Roberto C. Heros, MD

The world's neurological community lost recently one of its giants, C. Miller Fisher, who undoubtedly was one of the most influential neurologists of the 20th century. It is particularly fitting for neurosurgeons, particularly for those of us who have made a career of cerebrovascular surgery, to remember this remarkable man who was called "a neurosurgeon's neurologist" by my mentor, Robert G. Ojemann, when he introduced him as the 1983 Honored Guest of the Congress of Neurological Surgeons, the first non-neurosurgeon to have received that honor.

Dr. Fisher was born in the small town of Waterloo in Ontario, Canada in 1913. He did his undergraduate studies at the University of Toronto, where he also obtained his MD degree. While maintaining an outstanding academic record, he also excelled as a swimmer and water polo player. When he was a resident in internal medicine at the Royal Victoria Hospital in Montreal, World War II exploded and Dr. Fisher immediately volunteered and entered the navy as a medical officer. After a few months in England he was assigned to a cruiser of the Royal Navy, his ship was sunk and after nine hours in the water, he was rescued by a German ship and spent the next 3-1/2 years in a German prison camp. The story is told that it was there that he began his lifelong custom of not carrying a watch and disregarding time, an idiosyncrasy from which I suffered not infrequently when I asked him a clinical question while making late evening rounds in the wards of the Massachusetts General Hospital (MGH). Instead of giving the quick right answer, which he undoubtedly knew, Dr. Fisher would invite me to sit down and proceed with his characteristically Socratic way of leading me to the right answer, a process that could well take over an hour. Incidentally, Fisher was famous not only for this prolonged Socratic private educational lessons, but also for the manner in which, using kind sarcasm, he taught lessons that became impossible to forget. For example, again, during late evening rounds, I remember Dr. Fisher stopping me in the hall and saying, "I hear that you are going to operate tomorrow on a patient with a subclavian steal syndrome?" "And you're going to do that to prevent a stroke. Is that right?" When I answered yes, he went on to say, "And patients with subclavian steal syndrome have strokes. Is that right?" And then when I answered yes, of course, he went on to bring out his little white book and his pen and asked me to please give him the name of some of the patients that I had seen with a stroke from a subclavian steal syndrome so that he could look them up and study them carefully because for the last 30 years, he had been looking for such a patient and had never found one. This little dialogue took much longer than if he had said, "The subclavian steal syndrome never leads to stroke", a statement that I may have forgotten in short order.

After the War, Dr. Fisher came back to Canada and trained in neurology at the Montreal Neurologic Institute under Wilder Penfield's tutelage. After his neurologic training, he spent a year studying neuro-pathology at the Boston City Hospital under Dr. Raymond Adams and it is there that he became interested in cerebrovascular pathology and neurology. He then returned to the Montreal General Hospital where he made many of his

important early contributions over the next 4-1/2 years. At this time he was recruited by Dr. Adams, who had become Chief of Neurology at the MGH to return to Boston and develop the Stroke Service at the MGH where he spent the rest of his career. He could still be found on a daily basis in his late 80's working in his small crowded office at the MGH surrounded by piles of clinical records and pathologic slides. I was able to talk to him on the phone a few months before he died, at which time he was nearly blind, but still kept up with the recent literature using a loop; he remained acutely interested and could still comment eloquently on the latest developments in our field.

It is really impossible to even list, in a short written tribute to Dr. Fisher, his many contributions to neurology and neurosurgery. Rather, I will expand a bit more on the several monumental contributions that he made specifically to our field of cerebrovascular neurosurgery. Before expanding on that, I cannot but mention in passing just some of the other major contributions of Dr. Fisher to neurology. He was the first to describe in detail the subtleties of the neurologic examination of the comatose patient and in the process described phenomena such as ocular bobbing, reflex blepharospasm, ocular agitation, "wrong way eyes", pseudo sixth nerve palsy, etc. He described the syndromes of transient global amnesia and akinetic falling spells of the elderly. He made it possible to diagnose Jakob Creutzfeldt disease in life and characterized its typical myoclonic movements. He defined practically all of the well known lacunar strokes. He taught us that the lateral medullary syndrome was much more frequently due to vertebral occlusion than to occlusion of the posterior inferior cerebellar artery. Together with Drs. Hakim and Adams, he characterized the clinical syndrome of normal pressure hydrocephalus. He described late life migraine equivalents. He characterized the different clinical syndromes caused by hypertensive intracerebral hemorrhages and described the specific pathologic process, which he called lipohyalinosis, which leads to rupture of the small perforating arteries responsible for these hemorrhages. He recognized the phenomenon of "whiplash amnesia" and described the "Bell effect" in abulic patients. The way in which he recognized the latter phenomenon is characteristic of his lifelong learning from single clinical observations. Dr. Fisher was making his rounds on a patient that had severe abulia after being operated for an anterior communicating aneurysm. While at the bedside, he could not get the patient to speak at all, but then the telephone rang (hence the Bell's effect label for this phenomenon) and the patient picked up the telephone and responded appropriately with a couple of well enunciated sentences and then, again, stopped talking. Rather than ignoring this phenomenon, Dr. Fisher, who could not get the patient to talk to him at all at the bedside, went to the nurses station and called the patient on the telephone upon which the patient answered, "Oh hello Dr. Fisher, how are you...." And then quit talking again. Dr. Fisher's incredible volume of contributions during his long career are summarized in his upwards of 200 publications, several of them in the New England Journal of Medicine, of which, remarkably, over two thirds are single author publications!

Our field of cerebrovascular surgery would not be recognizable today were it not for Dr. Fisher's seminal contributions. He made the initial clinical pathologic correlation of thromboembolic stroke secondary to atherosclerosis at the origin of the cervical internal carotid artery (ICA), which led to the performance of carotid endarterectomies. He elucidated the mechanism of hemorrhagic stroke which he correctly attributed to transient embolic occlusion of a major cerebral artery, followed by spontaneous lysis of the clot and reperfusion injury on the distal territory. He described the term "transient ischemic attacks" and taught us that these attacks were frequently due to atherosclerosis at the origin of the ICA. He also led the early efforts to treat these patients prophylactically with anticoagulation. He described transient monocular blindness and again related it to severe stenosis at the origin of the ICA. He described border zone or watershed ischemia and infarction, secondary to ICA severe stenosis or occlusion. He advocated for the use of heparin and then warfarin for the prevention and attenuation of thromboembolic stroke. He told the world of neurology and cardiology that patients with atrial fibrillation should be treated prophylactically with Coumadin even if they had never had an embolic event. He presented this concept at one of the early Princeton conferences on cerebrovascular disease and when

asked by a prominent neurologist if he was aware of the fact that cardiologists did not advocate the prophylactic use of anticoagulation in patients with atrial fibrillation that had not had embolic events, Fisher responded something along the lines of "Of course I'm aware of that, why do you think I would be wasting my time and yours by talking about something that is already known?" Dr. Fisher described the clinical picture of cerebellar hemorrhage and told us of the need to operate on these patients urgently to save their lives and neurologic function. His confidence in making this diagnosis clinically was highlighted at one of the weekly brain cutting conferences at the MGH before the days of CT scanning. The patient had the typical syndrome of cerebellar hemorrhage, but when the story was told that the patient was operated and the surgeon did not find a hemorrhage, the other distinguished neurologists present all tried to come up with alternative diagnoses. Finally, when asked what he thought, Dr. Fisher said that it was too bad that the surgeon could not find the hemorrhage because undoubtedly that is what the patient had. Then the brain was cut and sure enough, the surgeon's track on the cerebellum stopped just short of the deep cerebellar hemorrhage that ultimately killed the patient.

Dr. Fisher described the "string sign" as characteristic of carotid dissection and taught us to treat dissections with anticoagulants rather than surgery which had frequently led to significant morbidity before we began to treat these patients with anticoagulants. As stated earlier, he characterized all the typical clinical syndromes caused by hypertensive intracerebral hemorrhages in different locations, which led us to diagnose and sometimes treat effectively some of these patients before the days of CT scanning. Finally, he clearly related vasospasm to the amount of blood in the basal cisterns on the initial CT scan which of course has led us to be able to predict with accuracy which patients will deteriorate later on from symptomatic vasospasm and to initiate measures to prevent or ameliorate such symptoms.

Perhaps the most remarkable human quality of this giant figure was his humility. I well remember how he always sat in the back of the room at the Ether Dome during our neurology/neurosurgery grand rounds while Dr. Raymond Adams always sat in the front row. A few years after I left the MGH, I was invited back to give grand rounds and I asked Dr. Fisher why the grand rounds had changed in format to didactic lectures as opposed to the former routine of presenting patients with mysterious neurologic syndromes and uncertain diagnoses. When these patients were presented, Dr. Fisher rarely spoke unless he was asked to and the "final word" as to what the patient had was generally given by Dr. Adams. When I asked Dr. Fisher why this format of patient-entered grand rounds had been discontinued, he told me that it was impossible to do that since Dr. Adams had retired and the only reason those rounds were effective was that Dr. Adams presided them and he (who incidentally was only a couple of years older than Dr. Fisher) was the last of the "classical neurologists" who knew all of neurology and who could analyze patients with any kind of neurologic problems. He then went on to say that he himself could not do this because, as opposed to Dr. Adams, he, Dr. Fisher, was an expert only at a few things in neurology and would look foolish trying to discuss patients with problems outside his limited field of knowledge. Such was the man that perhaps has done more than any other to influence the way we practice cerebrovascular surgery.

The changes in the treatment of patients with cerebrovascular disease. We encourage all neurosurgeons and cerebrovascular practitioners to become involved in our educational, research and advocacy activities. It is only through the efforts of dedicated individuals that we will continue to provide these important opportunities.

TECHNICAL FORUM: TYPE I SPINAL ARTERIOVENOUS FISTULAE (AVF)

BY KETAN R. BULSARA, MD

It was not too long ago that the mainstay of treatment for Type I spinal AVFs was microsurgical disconnection. As a matter of fact, it appeared so clear cut, that there was no need for discussion. With the advent of newer embolic agents and microcatheters, many centers have resorted to endovascular treatment as first line for these lesions. Though questions remain regarding the long-term durability of endovascular treatment for spinal AVFs, there is mounting evidence that it is an effective treatment option in carefully selected patients. For this technical forum, Dr. Adnan Siddiqui from the University of Buffalo and Dr. Gregory Zipfel from Washington University discuss their perspectives on the management of these lesions.

Surgical Management of Spinal Dural Arteriovenous Fistulas

Gregory J. Zipfel, MD

Introduction

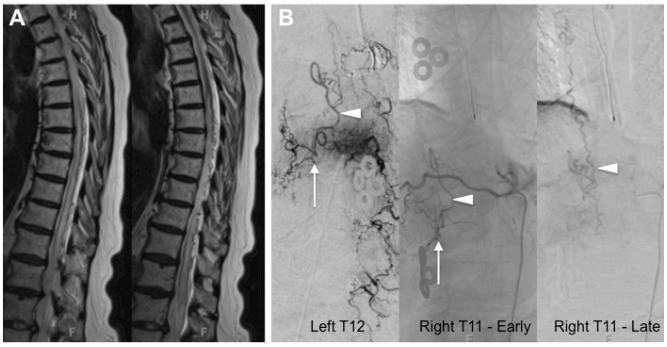
Spinal dural arteriovenous fistulas (SDAVFs) account for ~70% of spinal vascular malformations.1 They most commonly affect men (~80%) in their 50s and 60s,2 typically present with progressive myelopathy (often occurring in a stepwise fashion),3 and are readily cured via surgical or endovascular means. Best clinical outcomes occur in patients who are treated early, but substantial delays (> 1 year) between initial symptom onset and radiographic diagnosis is commonplace even in the modern era.3 This delay is ascribed to the subtle and often non-localizing nature of early symptoms, the frequent coexistence of additional spinal pathology such as degenerative spine disease, and the sometimes ambiguous or event absent findings on Magnetic Resonance Imaging (MRI). Once diagnosed and treated, however, the majority of SDAVF patients experience improvement or at least stabilization in their neurological condition, while only a minority suffers progressive neurological deficits. Long-term outcomes appear similar in patients treated via surgical or endovascular therapy, as long as complete obliteration of the fistulous site is obtained. Herein, the vascular anatomy, pre-operative imaging, intra-operative technique, and long-term outcomes following surgical management of SDAVFs are reviewed. The relative advantages and disadvantages of surgical vs. endovascular therapy for these uncommon but interesting vascular lesions are also discussed.

Vascular Anatomy

Though SDAVFs can occur anywhere along the spinal axis, they most commonly are located in the lower thoracic and lumbar regions – predominantly on the left vs. right side.4 These lesions are low-flow arteriovenous shunts between a radiculomeningeal artery and a radiculomedullary vein, and are usually located within the dural root sleeve at the intervertebral foramen. Radiculomeningeal arteries are branches of the segmental arteries that supply the dura at virtually every level along the spinal column. They are distinct from radiculomedullary arteries, which are also branches of segmental, but occur only intermittently along the spinal column to supply the anterior and posterior spinal arteries that perfuse the spinal cord. Radiculomedullary veins are also only intermittently found along the spinal column. These veins are usually found dorsolaterally within the intradural space and under normal conditions are the conduits by which venous outflow from the perimedullary venous plexus reaches the epidural space. In the pathological setting of a SDAVF, however, blood from a radiculomeningeal feeding artery is shunted into a radiculomedullary vein leading to venous hypertension, a decrease in the intramedullary arteriovenous gradient, reduced venous drainage from the spinal cord, and ultimately venous congestion and hypoxia within the spinal cord parenchyma.5 If left untreated, this pathophysiological process can eventually cause irreversible necrotizing myelopathy of the spinal cord – a condition known as Foix-Alajouanine syndrome.6

Pre-operative Imaging

MRI is the imaging modality of choice in patients with signs and/or symptoms of myelopathy. The primary radiographic findings suggesting the presence of a SDAVF are 1) a dilated perimedullary venous plexus, 2) hyperintense intramedullary signal on T2-weighted imaging, and 3) gadolinium-enhancement of the arteriovenous shunt within the intervertebral foramen.(Figure 1A) Of these, increased T2 signal change is the most sensitive radiographic finding.7 It is important to



note, however, that even in the absence of all three of these radiographic abnormalities, a SDAVF may be present. Therefore, in cases where a high index of suspicion exists, dedicated imaging of the spinal vasculature is still required.

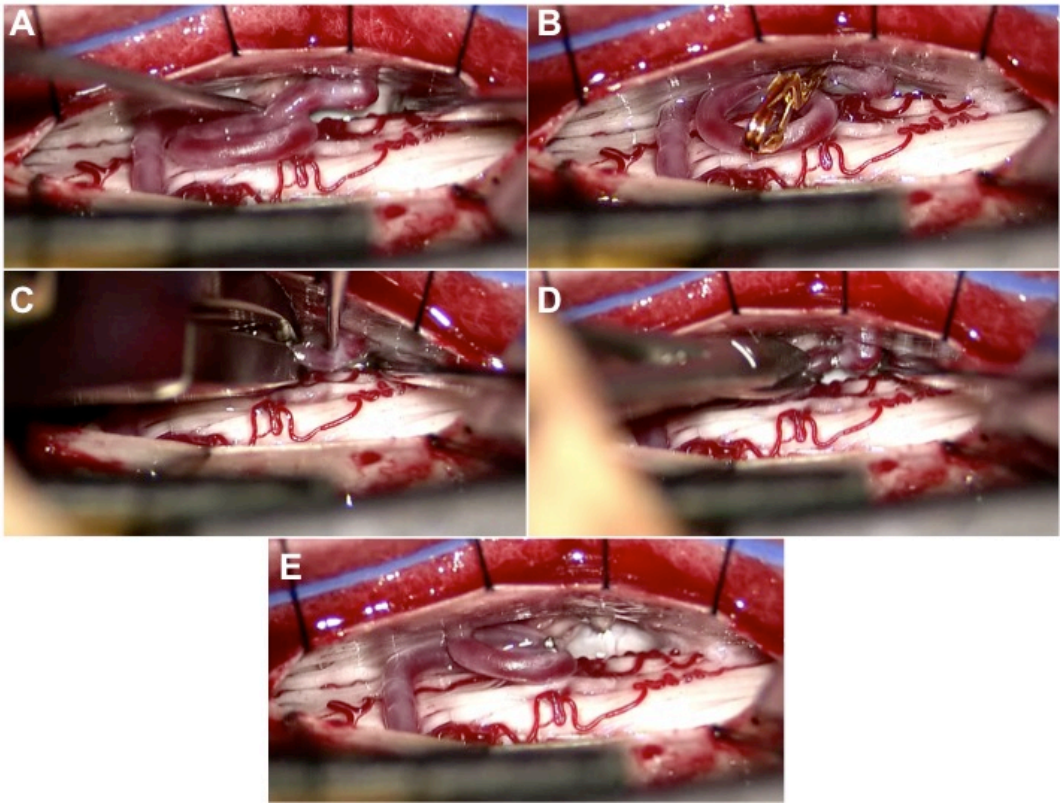
Gadolinium-enhanced Magnetic Resonance Angiography and contrast-enhanced Computed Tomographic Angiography are useful diagnostic adjuncts to spinal MRI. These imaging modalities can not only visualize a dilated perimedullary plexus, but are often able to localize the fistulous site itself and its associated arterialized radiculomedullary vein. By employing these non-invasive vascular imaging modalities, some have reported that the contrast load required during subsequent catheter spinal angiography can be reduced8 or the need for catheter spinal angiography can be altogether eliminated.4 However, many others believe that the potential for multiple radiculomeningeal feeders to a single SDAVF and the possibility for multiple SDAVFs (occurring in 2-3% of patients2) mandate catheter spinal angiography in all patients suspected of having a SDAVF.

Conventional catheter spinal angiography remains the gold standard imaging modality for the diagnosis of SDAVFs.(Figure 1A) Not only is it the most definitive means for identifying the fistula, but it is also critical in the pre-operative treatment planning and post-operative confirmation of

complete fistula obliteration. It is superior to non-invasive spinal vascular imaging due to its outstanding spatial resolution and its ability to delineate the location and relationship of the Artery of Adamkiewicz and other radiculomedullary arteries to the feeder(s) of the SDAVF – critical information when deciding whether endovascular therapy is a viable treatment option. Of course, the major drawback of catheter angiography is its associated risk; however, in the modern era, this risk appears exceedingly small.3

Surgical technique

Surgical treatment of SDAVFs is a straightforward technical procedure that carries an extremely high chance of cure. It involves a 1 or 2 level laminectomy, exposure of the involved intervertebral foramen, and intradural identification and disconnection of the arterialized radiculomedullary draining vein. Special anesthetic considerations include maintaining normal to high-normal mean arterial pressure throughout the procedure and careful positioning of the patient to avoid increased intra-abdominal pressure – both of which help maintain an adequate spinal arterial-venous gradient to avoid exacerbating the already fragile hemodynamic state within spinal cord. Somatosensory (SSEP) and motor (MEP) evoked potentials are typically employed to continuously monitor spinal cord function throughout the procedure. Precise identification of the spinal level of the fistula is a prerequisite for effective surgery. This is obtained via a combination of accurate pre-operative localization of the SDAVF in the angiographic suite and use of intra-operative fluoroscopy and/or neuronavigation. Once adequate exposure at the correct anatomic level is achieved, the spinal dura is opened in the midline and the arterialized draining vein is identified near the junction of the nerve root sleeve and spinal dura.(Figure 1B) Once identified, this arterialized vein is temporarily occluded with an aneurysm clip to assure stability of SSEP and MEP monitoring. (Figure 1C) The vein is then permanently disconnected via application of aneurysm clips, placement of hemoclips, or simple coagulation and division. (Figure 1D-F) No attempt is made to resect the dilated perimedullary venous complex. Coagulation of the epidural feeding arteries can also be performed, but this adjunctive



maneuver is not absolutely required. Intra-operative confirmation of SDAVF obliteration with catheter angiography is not routinely performed given the simplicity and overwhelming effectiveness of surgery as well as the technical difficulties associated with performing spinal catheter angiography in patients who are in the prone position. Indocyanine green videoangiography, on the other hand, is a simple intra-operative assessment tool for confirming completeness of SDAVF obliteration.9 Following surgery, it is strongly advocated to confirm radiographic cure of the fistula with a formal catheter spinal angiogram, given that the potential for further neurological injury is great when fistulas are incompletely treated.3

Surgical Results

The natural history of patients presenting with symptomatic SDAVFs is very poor, with 50% of patients becoming severely disabled (wheelchair-bound) within 3 years of symptom onset.10 Surgical obliteration of a symptomatic SDAVF clearly interrupts this process. In 2004, Steinmetz and colleagues published a meta-analysis examining the safety and efficacy of surgery (as well as endovascular therapy) for SDAVFs.11 They found that 98% of patients had complete obliteration of their fistula after surgery, and only 1.9% of patients suffered a significant peri-operative complication. The vast majority of these patients experienced improvement (55%) or stabilization (34%) of their presenting neurological deficits. Gait disturbances were more likely to improve following surgery than micturition disturbances. Similar results have recently been reported by Saladino and colleagues3 who documented surgical outcomes in 154 consecutive SDAVF patients – the largest single-institution surgical case series of SDAVF patients reported to date. It is important to note, however, that though major peri-operative complications are rare, 6-11% of patients suffer subjective or objective neurological decline in long-term follow-up after surgery.11 Given that this is most common in patients who are already severely affected by their SDAVF, this continued neurological deterioration despite complete fistula obliteration likely represents the consequences of late stage spinal cord dysfunction.

Surgical vs. Endovascular Treatment

No randomized controlled trials examining the efficacy of surgical vs. endovascular treatment for SDAVFs has been performed. Given the rarity of SDAVFs and the well-documented safety and efficacy of both surgical and endovascular therapy for these lesions, it is unlikely that such a trial will ever be completed. Therefore, treatment recommendations must be based on data culled from single-institution retrospective case series as well as one meta-analysis11 based on these primary reports. These data strongly suggest that SDAVF treatment with surgical and endovascular therapy is similarly safe, but that important differences in obliteration rates and long-term durability between the two treatment modalities exist. In the aforementioned meta-analysis, peri-operative complication rates were similar for surgery (1.9%) vs. endovascular therapy (3.7%).11 However, the rate of successful fistula obliteration with surgery was 98% vs. only 46% with endovascular therapy. 11 Importantly, the latter is likely an underestimate of the true obliteration rate of modern endovascular treatment of SDAVFs, given that it included case series in which older embolic agents such as polyvinyl alcohol were employed. When assessing modern case series in which liquid embolic agents (primarily N-butyl cyanoacrylate or NBCA) were exclusively utilized, obliteration rates of 60 to 90% are more commonly reported.12-17 Though initial experiences with the newest liquid embolic agent Onyx (ev3 Neurovascular, Irvine, CA) appear favorable,18-21 it remains to be determined whether improved SDAVF obliteration rates are achieved in larger case series. Regarding long-term treatment durability,

SDAVF recurrence following successful surgical obliteration is decidedly rare. With endovascular therapy, however, this is a well-documented phenomenon. In modern case series where liquid embolic agents such as NBCA were exclusively used, SDAVF recurrence occurs in 8 to 21% of patients.13, 16 Recurrence after Onyx embolization has also been documented.22

Conclusion

Patients harboring symptomatic SDAVFs have a poor natural history. Surgical management of these lesions is very safe, is highly effective at achieving complete fistula obliteration, and is highly durable in long-term follow-up. The vast majority of patients treated with surgery experience improvement or stabilization of their presenting neurological symptoms. Surgical intervention can therefore be considered a first line therapy in the majority of SDAVF patients. It can also be used as a secondary treatment option in patients where endovascular therapy has failed.

Figure Legends

An 85 year-old woman presented with 6 month history of progressive paraparesis and bowel/bladder incontinence. Spinal MRI revealed T2 hyperintensity within the thoracic and lumbar spinal cord as well as serpiginous vasculature within the dorsal spinal canal (Figure 1A). Spinal catheter angiography identified a SDAVF fed by the right T11 and left T12 segmental arteries (Figure 1B; arrows indicate site of fistula; arrowheads indicate arterialized perimedullary vein). The patient underwent T12 laminectomy to expose the arterialized perimedullary vein originating near the right T12 nerve root (Figure 2A). The draining vein was initially occluded with a temporary aneurysm clip to monitor stability of intra-operative SSEP and MEPs (Figure 2B). The draining vein was then occluded with hemoclips (Figure 2C,D) and divided with microscissors (Figure 2E,F). The patient experienced improvement in her motor symptoms immediately after surgery. Post-operative spinal catheter angiography revealed complete SDAVF obliteration.

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Endovascular management of Type I spinal dural vascular malformations

Maxim Mokin MD PhD1,4 and Adnan H Siddiqui MD

Type I spinal dural arteriovenous fistula (DAVF) is the most commonly encountered vascular malformation affecting the spinal column. The arterial supply to these fistulae originates from an aortic segmental branch feeding the nerve root, called the radicular artery. This lies adjacent to the radicular vein and – typically in an acquired fashion – develops an abnormal direct connection to the radicular vein. The ensuing venous hypertension results in retrograde transmission of venous hypertension to the epidural plexus alone which, secondary to its capacitance, may remain asymptomatic or to the longitudinal radiculomedullary veins where it leads to venous hypertension of the spinal cord and resultant myelopathy.

In recent years, endovascular embolization of type I fistulas has become increasingly utilized, largely because of the availability of newer liquid embolic agents, such as Onyx (ev3/Covidien) and N-butyl cyanoacrylate (NBCA). Significantly higher embolization rates are obtained with these newer agents than with the older particle embolization agents, such as polyvinyl alcohol, or coils. Patsalides et al.1 recently reviewed outcomes in patients treated with an endovascular approach and found the rates of successful embolization resulting in cure to be as high as 90%.

When compared to surgical obliteration of the fistula, endovascular treatment offers a markedly less-invasive approach. Diagnostic angiography with direct catheterization of segmental arteries is almost always used for evaluation of DAVF because it allows precise identification of the spinal level marking the exact site of the fistula. In straightforward cases, both the diagnostic study and the treatment can be performed during the same session. Many institutions now consider endovascular embolization as the initial treatment of choice. If selective catheterization of the fistula site is not possible or there is associated supply to the spinal cord from the artery of Adamkiewicz, open surgical intervention might be necessary.

We typically use a 5- or 6-French guide catheter to engage the ostia of the aortic segmental branch. The patient is heparinized to an activated coagulation time of 250 seconds. The microcatheter used depends on the exact agent planned because Onyx (for example) requires a dimethyl sulfoxide-compatible catheter. The microcatheter is navigated over a microwire as far distally as possible, ideally up to or even through the fistulous connection. This is best seen where there is an abrupt change in the diameter from a small caliber artery to a dilated vein. At this point, we perform provocative testing with sodium amytal and lidocaine to test for collateral arterial supply to eloquent nervous tissue. If this is negative, we proceed with embolization. NBCA polymerizes immediately after it is injected from the microcatheter upon contact with blood and thus has limited migration properties within the vessel lumen. Because of this feature, the microcatheter tip has to be positioned directly at the site of the fistula. In contrast, Onyx is a more recently developed nonadhesive liquid embolic agent that, once injected, can migrate more distally and thus cause embolization of deeper fistula sites that are not accessible even with the smallest microcatheters. A case of Onyx embolization is shown in Figure 1.

In conclusion, the best treatment approach (endovascular versus surgical) is the one that provides both safe and definitive embolization of the fistula. Meticulous examination of a diagnostic spinal angiogram ensures a correct understanding of the vascular supply to the fistula and appropriate selection of patients for endovascular intervention. Utilization of modern liquid embolic agents results in high obliteration and low recurrence rates, which are necessary to achieve successful and long-term clinical improvement.

Legend

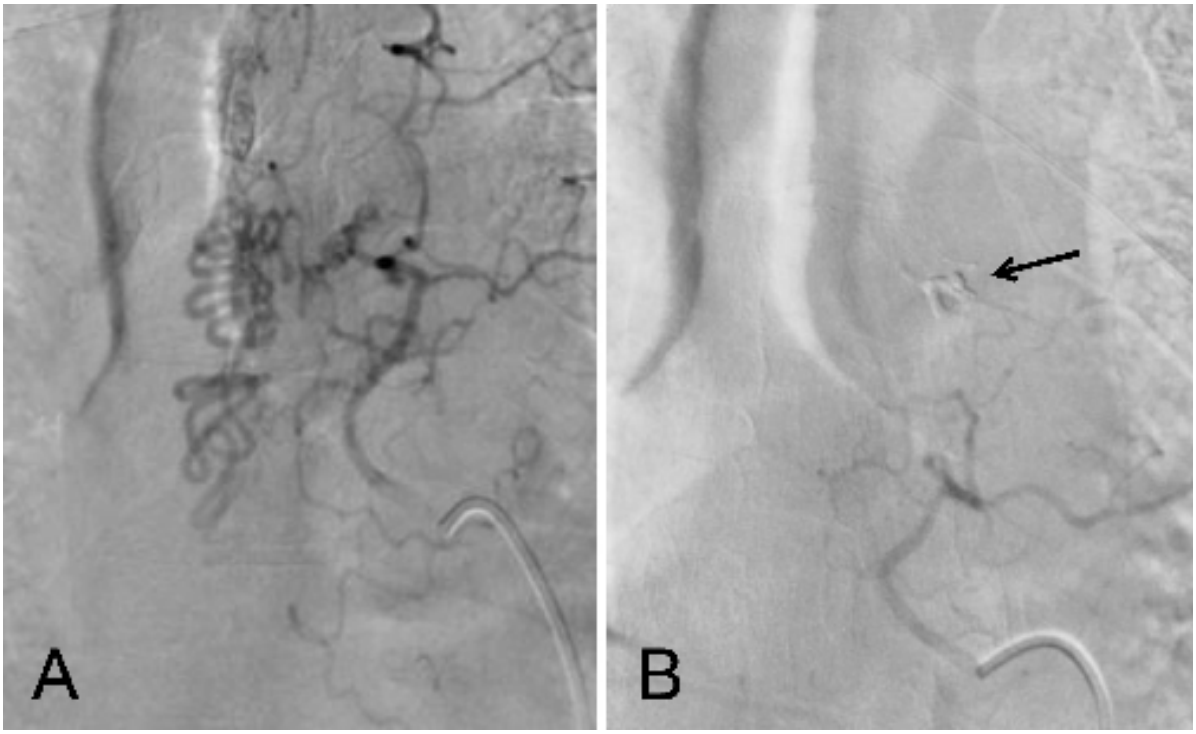

Figure 1. A) Spinal angiogram showing dural arteriovenous fistula supplied by the left T6 radicular branch. B) Following successful embolization of the left T6 pedicle with Onyx (ev3/Covidien), no residual filling of the fistula can be visualized. Arrow is pointing to the Onyx cast.

Reference

1. Patsalides A, Santillan A, Knopman J, Tsiouris AJ, Riina HA, Gobin YP: Endovascular management of spinal dural arteriovenous fistulas. J Neurointerv Surg 3:80-84, 2011

TREASURER'S MESSAGE

Brian Hoh, MD, PhD

Despite difficult economic times, the JCVS was financially solvent in FY 2012. The JCVS has been able to carry out its mission while remaining fiscally responsible. We were able to hold a successful annual meeting, residents' course, and fellows' course. The JCVS sponsored two Robert J. Dempsey Resident Research Awards for $15,000 each to two neurosurgery residents conducting cerebrovascular research. The JCVS contributed $10,000 to the Washington Committee, and $20,000 to support the NREF/Cerebrovascular Section Research Grant. This year, the JCVS also donated $15,000 to a fund for Ron Englebreit's family and $5000 to the Brain Aneurysm Foundation for the Christopher Getch research grant.

The 2013 Joint AANS/CNS Cerebrovascular Section Annual meeting will be held in Honolulu, Hawaii from February 3-5th. This year's meeting is in partnership with the Society for Neurointerventional Surgeons (SNIS). As in previous years, our meeting will immediately precede the International Stroke Conference. This year, we also welcome and look forward to the participation from the Joint Mt. Bandai Symposium Neuroscience/Pan-Pacific Neurosurgery Conference attendees at our annual meeting.

The meeting will be held at Sheraton Waikiki Hotel. Registration will open in October. The anticipated opening of the abstract center is first week of September

OPPORTUNITIES FOR FUNDING

AANS FELLOWSHIP/GRANTS

http://www.aans.org/Grants%20and%20Fellowships.aspx

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Calendar

October 6-10, 2012

CNS Annual Meeting

Chicago, Il

February 3-5, 2013

Cerebrovascular Section Meeting

Honolulu, Hawaii

February 15-17, 2013

North American Skull Base Society Meeting

Miami, Florida

April 27-May 1, 2013

AANS Annual Meeting

New Orleans, Louisiana