

Intensive Care Unit Management of Interventional Neuroradiology Patients

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The management of interventional neurologic patients in the intensive care unit (ICU) is based on their underlying disease for the most part. Patients with ischemic stroke are largely managed like patients with ischemic stroke who have not undergone interventional procedures, and the same is true for those with an aneurysmal subarachnoid hemorrhage or intracerebral hemorrhage (ICH) secondary to an arteriovenous malformation, for example. Having said this, there are some special considerations that require special mention when it comes to managing patients after catheter-based procedures. Unfortunately, the data supporting these management techniques are sparse and generally of low quality. As such, much of what follows is based on less than class I data and should be viewed as no more than informed opinion.

Aneurysmal subarachnoid hemorrhage

Since the US Food and Drug Administration (FDA) approved Guglielmi detachable coils in 1995, more than 100,000 patients have had their aneurysm coiled, many of these after subarachnoid hemorrhage [1]. With the publication of the International Subarachnoid Aneurysm Trial

(ISAT) trial results in 2002, there has been an increasing shift in many centers to coil all but the most anatomically unfavorable ruptured aneurysms [2]. Despite these developments, little has been written about the specific management of these patients, leading one to believe that management of clipped and coiled patients is identical. Although the basics, such as immediate blood pressure stabilization, fluid management, use of nimodipine, ventricular drainage, and angioplasty when needed, are somewhat standardized [1], unique situations do arise with endovascularly treated patients that result in minor alterations.

Anticonvulsant drug use

Over the past few years, there is a growing realization that the risks of routine anticonvulsant drug (AED) use, especially phenytoin, probably outweigh the benefits [3]. As a result, many have advocated loading patients with AEDs only until the aneurysm is secured [4]. Others believe that comatose patients might benefit in the acute setting until a nonconvulsive status is ruled out [5]. Still others believe that patients who have undergone significant cerebral resections or have extensive cortical injury secondary to ICH or subdural hematoma should receive prophylaxis at least until the risk of vasospasm has passed [6]. Although no class I data actually exist for this patient population, we tend to use AEDs on a perioperative basis

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in surgically treated patients but avoid their use in coiled patients.

As more data accumulate regarding the utility and risk of perioperative AEDs, differences between the management of coiled and clipped patients may disappear.

Intraventricular tissue plasminogen activator for casted ventricles

Increasingly, it has been shown that intraventricular blood is associated with a worse outcome after aneurysmal subarachnoid hemorrhage [7]. In some cases, external ventricular drain patency is threatened, leading to elevations in intracranial pressure (ICP); in other cases, massive clot burden leads to prolonged drain dependency and an increased risk of ventriculitis [8]. For these reasons, several investigators have begun to use intraventricular thrombolysis in select patients [9,10]. Although surgically clipped lesions seem to be undisturbed by these proteases, considerable debate exists with regard to lesions that have been coiled [9,11]. Although the fear seems to be more theoretic than experiential, the thought of dissolving the dome of a lesion and thereby gaining access to clot within the coil interstices continues to give many clinicians reason to pause. This fact, combined with the ability of the surgeon performing a pterional craniotomy to open the lamina terminalis with little additional effort or morbidity, has led some to advocate craniotomy over coiling in cases of casted ventricles [12].

External ventricular drainage

Although the use of external ventricular drainage in patients with subarachnoid hemorrhage is quite varied from institution to institution, there is a general feeling that patients in a coma (Hunt and Hess grades 4 and 5) benefit, as do those in better condition who have ventriculomegaly and deteriorating findings on examination [13]. Although patients in a coma are generally immediately drained to exclude increased ICP as a cause of their coma, many awake patients with ventriculomegaly, even with deteriorating examination results, are taken to the operating room before ventriculostomy and managed by other techniques, such as spinal drainage, in an effort to avoid rebleeding precipitated by changes in transmural pressure [14]. This approach still allows for placement of a ventricular drain through the craniotomy opening should the relaxation be insufficient

and has allowed several groups to report anecdotal reductions in the incidence of ventricular drainage and, by extension, ventriculitis. Still others have advocated leaving the spinal drain in after surgery, because anecdotal evidence may suggest that this leads to a diminished incidence of symptomatic vasospasm, presumably by facilitating the clearance of subarachnoid blood [15].

In contrast, endovascularly treated patients usually have drains placed before coiling if there is any ventriculomegaly whatsoever. The reason for this is twofold. First, patients who may be alert before surgery may develop increases in ICP when laid flat on the angiography table. Second, if there is any untoward event during angiography, such as might occur with aneurysm dome perforation, the delay in putting in a drain may be significant. Furthermore, if the coiling is uncomplicated but the patient experiences some major thromboembolic complication (eg, dissection, lost coil or guidewire, parent vessel thrombosis, catheter embolus), full-dose anticoagulation may be necessary. If the patient subsequently develops worsening hydrocephalus requiring drain placement, the risk of catheter-induced ICH is significant.

Antiplatelet agents

Generally speaking, patients with aneurysmal subarachnoid hemorrhage managed with craniotomy are never placed on antiplatelet agents, even if they have relatively strong indications for being on them, for fear of postoperative subdural or epidural bleeding. In addition, many patients require multiple additional invasive procedures, such as ventriculostomy, gastrostomy, tracheostomy, and central line placement. With the advent of the Neuroform stent and its use in the treatment of wide-necked aneurysms, the issue of antiplatelet therapy, particularly aspirin together with clopidogrel, has resurfaced. For the most part, it is the general feeling that when wide-necked aneurysms must be coiled in the setting of subarachnoid hemorrhage, it is better to perform the procedure with a balloon-assisted technique rather than place a stent if at all possible [16]. Residua can be dealt with later by delayed stent placement after the need for additional procedures has passed. In the case that a patient is on aspirin and clopidogrel because of stent placement, it may be feasible to reverse these drugs temporarily by fresh single-donor platelet transfusion, followed by reinstitution of the drugs after an undetermined period of watchful waiting.

Antifibrinolytic therapy

Although early trials showed this approach to reduce rebleeding at the expense of increased delayed ischemia with no improvement in overall outcome [17], recent data may suggest that ultra-early use (first 48 hours after bleeding) while the patient is being transported for definitive treatment may actually improve the outcome without increasing delayed ischemia [18]. Although those performing open craniotomy have been quick to support such an approach, especially given the evidence that immediate surgery is often less than immediate, there has been some concern among endovascular surgeons that such therapy might lead to an increase in the thromboembolic complications associated with coiling. Although there is no evidence to date that this is the case, careful assessment may require an independent assessment of the risk of these therapies in endovascularly managed patients.

Vasospasm monitoring and treatment

Although there are reports suggesting an increased, decreased, and similar incidence of vasospasm in patients managed endovascularly compared with those managed via craniotomy, there is no evidence that the medical management of coiled patients should be any different [19]. Nimodipine therapy and intravascular volume maintenance as well as the use of hypertonic saline, mannitol, and pressors for those with symptoms are essentially the same for all [1]. The early use of angioplasty and intra-arterial vasodilators in patients who fail to respond to medical maneuvers is also the same. Although part of the medical management of these patients includes optimization of blood oxygen-carrying capacity and rheology and, by extension, attention to marked anemia, most patients undergoing clipping have also undergone catheter angiography and are therefore also at risk for retroperitoneal hemorrhage. Thus, any patient with unexplained blood loss should undergo abdominal and pelvic CT scanning to rule this out. After catheter-based procedures, those patients may be at increased risk because of the use of larger guiding catheter sheaths.

Partially treated aneurysms

As long as the site of aneurysmal bleeding has been excluded from the circulation, there is little risk for acute rebleeding during the ICU stay (7–21 days). In one study in which 80% of patients

were undertreated and heparin was used to guard against a thromboembolic phenomenon, there were no episodes of early rebleeding even when induced hypertension was used to treat spasm [20]. Although it is often difficult to know the exact site of the bleeding, and one can think of exceptions, patients with partially coiled aneurysms can be treated the same as those with perfectly coiled or clipped lesions. Additional unruptured aneurysms have been shown to remain unruptured in the setting of even aggressive triple-H therapy or balloon angioplasty and do not require treatment in the acute setting [21]. Occasionally, a coil may become stretched as the coiling is coming to a conclusion, leaving a tail in the efferent or afferent vessel. Usually, this requires no additional management, but if there is associated thromboembolism, aspirin (81 mg/d) is usually prescribed and platelet transfusions are used for significant invasive procedures.

Ischemic stroke

Patients with ischemic stroke are rarely transferred to the ICU unless their mental status is compromised. As a result, most of the ischemic strokes that are managed are caused by large-vessel thromboembolism and are being treated as part of acute stroke protocols. These protocols include the FDA-approved use of intravenous tissue plasminogen activator (rt-PA) for those presenting within 3 hours as well as the non-FDA-approved use of intra-arterial rt-PA or urokinase within 6 hours [22–24]. For those who receive intravenous rt-PA but do not recanalize, “bridging protocols” have been developed to allow for the subsequent use of intra-arterial “lytics” within 6 hours. For those who still do not recanalize or for those presenting with large areas of perfusion-diffusion “mismatch” on perfusion-weighted imaging or diffusion-weighted imaging MRI, the FDA has approved the use of a mechanical endovascular corkscrew [25,26]. This has been used out to 8 hours in the anterior circulation with varied success and out to 12 to 18 hours in the posterior circulation. In terms of ICU management, patients are initially kept hypertensive until recanalization is achieved and their pressure is then lowered. Ideally, patients are managed with a combination of anticoagulation and antiplatelet therapy (aspirin and clopidogrel) unless the infarct area is greater than 30 to 40 mL. With these larger infarcts, especially when reperfusion is successful, we have generally gone with

antiplatelet therapy alone for fear of inducing hemorrhagic conversion of the infarcted tissue.

Patients who ultimately develop large hemispheric strokes have been treated with surgical decompression as well as aggressive medical therapy depending on family desires [27]. The early data from the small but randomized, prospective, multicenter trial for hemicraniectomy seem to suggest a survival advantage but no improvement in function [28]. Moreover, hemicraniectomy is not benign and has been associated with a whole host of side effects that are difficult to manage, such as hydrocephalus, wound breakdown, meningitis, and even delayed epidural bleeding. Patient selection for this procedure is therefore key [29]. Meta-analysis suggests that younger patients, especially those with massive swelling despite incomplete middle cerebral artery territory infarction, specifically that involving the nondominant hemisphere, may be best served [30]. For others, we have used a combination of mild (34.5°C–35.5°C) prolonged (7–10 days) hypothermia together with hypertonic saline (2% and 3%), followed by gradual withdrawal of both [31]. Dramatic anecdotal successes exist, but there are an equally large number of patients who have not responded.

General

For patients with hemorrhage as well as ischemic stroke, significant efforts are made to maintain normal magnesium and glucose levels, with frequent testing and intravenous drips when necessary. These efforts are based on strong data for magnesium in animal models as well as in human beings with eclampsia, neonatal birth injury, and subarachnoid hemorrhage [32]. The data for normoglycemia are class I but were acquired in patients with a whole host of injuries (not just those to the nervous system) [33]. In addition, although hypothermia is used experimentally in the ICU, normothermia and the avoidance of fever are generally considered a “good idea” for all neurologically injured patients [34]. This comes from the fact that hyperthermia is closely associated in multivariate models with a poor outcome after ischemia and hemorrhagic stroke [34]. As a result, patients are managed with a variety of surface-cooling devices (eg, blankets, pads) until such time that failure necessitates intravascular cooling with a variety of FDA-approved and non-FDA-approved devices. Although studies have not randomized patients to

device-maintained normothermia or placebo, such studies are currently being organized and should provide ample support for these efforts going forward [34,35].

Summary

The ICU management of patients undergoing endovascular procedures is complex and varied. It depends not only on the underlying disease process but on the exact nature of the procedure performed, the procedure's success, and whether or not there were any procedural complications, however minor. As a result, there are few class I data to support much of what is done. In fact, most management techniques are drawn from other settings and anecdotal experience. We have highlighted just a few of the issues that arise.

As an increasing number of endovascularly treated patients find their way into ICUs, it should be increasingly important to examine whether the treatments used are ideal and, if not, how they can be improved on. This is likely to require multicenter collaboration as well as a change in the way society views the neurologically injured patient. Currently, many states prohibit the enrollment of comatose patients into randomized clinical trials because they do not recognize next of kin assent as sufficient. We can only hope that as the debate rages over quality in health care, comatose patients are also allowed to participate in the fruits of evidenced-based approaches.

References

- [1] Wijdicks EF, Kallmes DF, Manno EM, et al. Subarachnoid hemorrhage: neurointensive care and aneurysm repair. *Mayo Clin Proc* 2005;80:550–9.
- [2] Molyneux A, Kerr R, Stratton I, et al. International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms: a randomised trial. *Lancet* 2002;360:1267–74.
- [3] Naidech AM, Kreiter KT, Janjua N, et al. Phenytoin exposure is associated with functional and cognitive disability after subarachnoid hemorrhage. *Stroke* 2005;36:583–7.
- [4] Baker CJ, Prestigiacomo CJ, Solomon RA. Short-term perioperative anticonvulsant prophylaxis for the surgical treatment of low-risk patients with intracranial aneurysms. *Neurosurgery* 1995;37:863–70.
- [5] Claassen J, Peery S, Kreiter KT, et al. Predictors and clinical impact of epilepsy after subarachnoid hemorrhage. *Neurology* 2003;60:208–14.
- [6] Claassen J, Mayer SA, Kowalski RG, et al. Detection of electrographic seizures with continuous

- EEG monitoring in critically ill patients. *Neurology* 2004;62:1743–8.
- [7] Claassen J, Bernardini GL, Kreiter K, et al. Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: the Fisher scale revisited. *Stroke* 2001;32:2012–20.
 - [8] Lozier AP, Sciacca RR, Romagnoli MF, et al. Ventriculostomy-related infections: a critical review of the literature. *Neurosurgery* 2002;51:170–81.
 - [9] Findlay JM, Jacka MJ. Cohort study of intraventricular thrombolysis with recombinant tissue plasminogen activator for aneurysmal intraventricular hemorrhage. *Neurosurgery* 2004;55:532–7.
 - [10] Naff NJ, Hanley DF, Keyl PM, et al. Intraventricular thrombolysis speeds blood clot resolution: results of a pilot, prospective, randomized, double-blind, controlled trial. *Neurosurgery* 2004;54:577–83.
 - [11] Azmi-Ghadimi H, Heary RF, Farkas JE, et al. Use of intraventricular tissue plasminogen activator and Guglielmi detachable coiling for the acute treatment of casted ventricles from cerebral aneurysm hemorrhage: two technical case reports. *Neurosurgery* 2002;50:421–4.
 - [12] Findlay JM, Weir BK, Kassell NF, et al. Intracisternal recombinant tissue plasminogen activator after aneurysmal subarachnoid hemorrhage. *J Neurosurg* 1991;75:181–8.
 - [13] Le Roux PD, Elliott JP, Newell DW, et al. Predicting outcome in poor-grade patients with subarachnoid hemorrhage: a retrospective review of 159 aggressively managed cases. *J Neurosurg* 1996;85:39–49.
 - [14] Connolly ES Jr, Kader AA, Frazzini VI, et al. The safety of intraoperative lumbar subarachnoid drainage for acutely ruptured intracranial aneurysms: technical note. *Surg Neurol* 1997;48:338–42.
 - [15] Klimo P Jr, Kestle JR, MacDonald JD, et al. Marked reduction of cerebral vasospasm with lumbar drainage of cerebrospinal fluid after subarachnoid hemorrhage. *J Neurosurg* 2004;100:215–24.
 - [16] Han PP, Albuquerque FC, Ponce FA, et al. Percutaneous intracranial stent placement for aneurysms. *J Neurosurg* 2003;99:23–30.
 - [17] Kassell NF, Torner JC, Adams HP Jr. Antifibrinolytic therapy in the acute period following aneurysmal subarachnoid hemorrhage. Preliminary observations from the Cooperative Aneurysm Study. *J Neurosurg* 1984;61:225–30.
 - [18] Hillman J, Fridriksson S, Nilsson O, et al. Immediate administration of tranexamic acid and reduced incidence of early rebleeding after aneurysmal subarachnoid hemorrhage: a prospective randomized study. *J Neurosurg* 2002;97:771–8.
 - [19] Hohlrieder M, Spiegel M, Hinterhoelzl J, et al. Cerebral vasospasm and ischaemic infarction in clipped and coiled intracranial aneurysm patients. *Eur J Neurol* 2002;9:389–99.
 - [20] Bernardini GL, Mayer SA, Kossoff SB, et al. Anti-coagulation and induced hypertension after endovascular treatment for ruptured intracranial aneurysms. *Crit Care Med* 2001;29:641–4.
 - [21] Swift DM, Solomon RA. Unruptured aneurysms and postoperative volume expansion. *J Neurosurg* 1992;77:908–10.
 - [22] National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995;333:1581–7.
 - [23] del Zoppo GJ, Higashida RT, Furlan AJ, et al. PROACT: a phase II randomized trial of recombinant pro-urokinase by direct arterial delivery in acute middle cerebral artery stroke. PROACT Investigators. *Stroke* 1998;29:4–11.
 - [24] Wardlaw JM. Overview of Cochrane thrombolysis meta-analysis. *Neurology* 2001;57(Suppl):S69–76.
 - [25] Becker KJ, Brott TG. Approval of the MERCI clot retriever: a critical view. *Stroke* 2005;36:400–3.
 - [26] Gobin YP, Starkman S, Duckwiler GR, et al. MERCI 1: a phase I study of mechanical embolus removal in cerebral ischemia. *Stroke* 2004;35:2848–54.
 - [27] Mayer SA, Kossoff SB. Withdrawal of life support in the neurological intensive care unit. *Neurology* 1999;52:1602–9.
 - [28] Fandino J, Keller E, Barth A, et al. Decompressive craniotomy after middle cerebral artery infarction. Retrospective analysis of patients treated in three centres in Switzerland. *Swiss Med Wkly* 2004;134:423–9.
 - [29] Curry WT Jr, Sethi MK, Ogilvy CS, et al. Factors associated with outcome after hemispherectomy for large middle cerebral artery territory infarction. *Neurosurgery* 2005;56:681–92.
 - [30] Gupta R, Connolly ES, Mayer S, et al. Hemispherectomy for massive middle cerebral artery territory infarction: a systematic review. *Stroke* 2004;35:539–43.
 - [31] Georgiadis D, Schwarz S, Aschoff A, et al. Hemispherectomy and moderate hypothermia in patients with severe ischemic stroke. *Stroke* 2000;33:1584–8.
 - [32] van den Bergh WM. Magnesium sulfate in aneurysmal subarachnoid hemorrhage. A randomized controlled trial. *Stroke* 2005.
 - [33] van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;345:1359–67.
 - [34] Mayer SA, Kowalski RG, Presciutti M, et al. Clinical trial of a novel surface cooling system for fever control in neurocritical care patients. *Crit Care Med* 2004;32:2508–15.
 - [35] Mayer S, Commichau C, Scarmeas N, et al. Clinical trial of an air-circulating cooling blanket for fever control in critically ill neurologic patients. *Neurology* 2001;56:292–8.