

A primer of neurologic emergencies: summary from the American Thoracic Society Meeting 2016

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At the American Thoracic Society Meeting in 2016, there were a number of interesting sessions focused on neurological critical care. Tom Bleck, MD provided a lecture, some portion of which is summarized here. In addition we have added information from other talks and recent publications in this area.

Ischemic infarct

As compared with placebo, intravenous alteplase [IV tissue plasminogen activator (tPA)] administered between 3 and 4.5 hours after onset of symptoms significantly improved clinical outcomes in patients with acute ischemic stroke; tPA was more frequently complicated by symptomatic intracranial hemorrhage (1). Regardless of whether IV tPA is administered, in patients with anterior circulation proximal arterial occlusion seen on angiography, a small infarct core, and moderate-to-good collateral circulation, intra-arterial therapy is safe and can be considered (2). In patients undergoing endovascular thrombectomy, studies support decreased mortality and improved functional outcomes (3,4). Early IV magnesium sulfate therapy does not improve outcomes in ischemic stroke (5).

In patients with malignant edema, surgical decompression can be considered within 48 hours of stroke onset potentially to decrease mortality and poor outcome. However, this decision should be one that is made with an emphasis on the patient's previously understood wishes related to survival and dependency (6).

Intracerebral hemorrhage

There is a trend towards improved functional outcomes

with SBP <140 mmHg in INTERACT II (7), though no difference in functional outcome or mortality was seen in ATACH-2 with aggressive SBP management <140 mmHg (8). 3-factor or 4-factor PCC should be administered rather than FFP to patients with vitamin-K antagonist-associated intracranial hemorrhage and INR >1.4 (9).

There is no difference in functional outcome with craniotomy for supratentorial hemorrhage, though there may be a small survival benefit in superficial intracerebral hemorrhage without intraventricular blood (10). Minimally invasive surgery with instillation of tPA decreases hematoma volume and perihematoma edema (11); it is yet unknown whether this approach affects outcomes, but there is an active trial underway. In patients with large intraventricular hemorrhage and small intracerebral hemorrhage meeting appropriate criteria, there was a reported mortality benefit following protocolized administration of intraventricular tPA (CLEAR III *preliminary* results presented at International Stroke Conference, Los Angeles, 2016).

Aneurysmal subarachnoid hemorrhage (SAH)

Nimodipine should be administered to patients with aneurysmal SAH (12). The addition of statins was not associated with reduction in vasospasm or improvement in outcomes (13). Intravenous magnesium sulfate does not improve clinical outcome after SAH, and therefore routine administration cannot be recommended (14).

Aneurysmal SAH is often complicated by seizures; among patients treated with phenytoin (PHT), burden of exposure to PHT predicts poor neurologic and cognitive outcome after SAH (15).

When symptomatic vasospasm or delayed cerebral ischemia is diagnosed, the initial treatment is hemodynamic augmentation and maintenance of euvolemia. While there are no randomized trials of this intervention, expert consensus is in favor (16).

Traumatic brain injury

In adults with severe diffuse traumatic brain injury, refractory intracranial hypertension is an unfortunate complication. Although early bifrontotemporoparietal decompressive craniectomy decreases intracranial pressure and ICU length of stay, it is associated with more unfavorable outcomes (17). Additionally, therapeutic hypothermia in combination with standard care does not result in better outcomes than with standard care alone (18).

Status epilepticus

A retrospective study of continuous electroencephalography (cEEG) in the medical intensive care unit (MICU) revealed that—especially in patients with sepsis—non-convulsive electrographic seizures and periodic epileptiform discharges were frequent. Both seizures and periodic discharges were associated with poor outcome (19).

For subjects in status epilepticus, intramuscular midazolam is at least as safe and effective as intravenous lorazepam for prehospital seizure cessation (20).

Cardiac arrest

Following cardiac arrest of presumed cardiac etiology, there is evidence to support improved neurological outcomes with mild hypothermia to 32–34 °C (21). However, in a more recent study, unconscious survivors of out-of-hospital cardiac arrest who underwent temperature management with a target temperature of 36 °C had similar mortality and functional outcomes as those with a target temperature of 33 °C (22).

In the era of post-cardiac arrest temperature management, prognostication can be challenging. Multimodal prognostication provides the most reliable prognostication of poor outcome, though prediction of good prognosis remains inaccurate. It is possible to achieve 100% positive predictive value of poor neurological outcome using the combination of absence of EEG reactivity, incomplete recovery of brainstem reflexes in normothermia, and elevated neuron-specific enolase (23).

Summary

In summary, the field of neurological critical care has evolved in recent years. A robust body of evidence is being developed to help guide intensivists regarding optimal management of patients. Only with further research will new therapeutic approaches emerge over time.

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Footnote

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