Myocardial and Vascular Management in Neurosurgical Critical Care

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Neurogenic Stunned Myocardium

Overview

The most likely etiology of cardiac injury after SAH is the increased release and decreased reuptake of catecholamines, specifically circulating norepinephrine. Neurogenic cardiac injury following SAH is characterized by electrocardiographic abnormalities (ECG), arrhythmias, myocardial infarction (both non-ST elevation and ST elevation), left ventricular dysfunction, elevation of troponin, and cardiac arrest. Stress-induced cardiomyopathy, also called "neurogenic stunned myocardium," "transient left ventricular apical ballooning," "Takotsubo cardiomyopathy," and "broken heart syndrome," is an increasingly reported phenomenon. It is a transient condition and is typically precipitated by intense physiologic stress including that precipitated by brain injury and has even been described in the context of a profound emotional crisis. Typically stress-induced cardiomyopathy is reversible after several weeks.

Prevention

Prevention is by avoiding the physiologic stress itself. All patients presenting with intracranial pathology should have a 12-lead ECG and cardiac enzyme measurements (e.g., troponin) on admission and telemetry until their neurologic condition has stabilized. A thorough history and physical examination is necessary to identify patients at risk for primary cardiac disease. Awareness of the potential for neurogenic stunned myocardium, especially in those patients presenting with severe neurologic injury, will aid in the prompt search and diagnosis if myocardial stunning occurs.

Diagnosis

The clinical presentation is identical to that of an acute MI; however, coronary arteriography shows no critical lesions. It is important to differentiate the two; stunned myocardium is a reversible condition that will resolve completely in about 80% of the patients within days-weeks after the initial event, whereas a primary ischemic injury may cause irreversible cardiac dysfunction. Other diagnostic characteristics of neurogenic myocardial stunning include:

- Abnormal wall motion involving the cardiac apex and midportion with relative sparing of the base, termed "apical ballooning"
- ST segment elevation or depression or T-wave changes
- A prolonged QT interval
- Increased cardiac enzymes
- More common in elderly or postmenopausal females
- Precipitated by acute physiologic or emotional stress

Characteristics that are commonly associated with neurogenic myocardial stunning and not with ischemic acute coronary syndrome (ACS) are no history of cardiac problems, new onset left ventricular dysfunction, cardiac wall motion abnormalities on echo that do not correlate with the coronary vascular distribution, and cardiac troponin levels <2.8 ng/ml. If there is doubt, then coronary angiography should be performed if feasible.

Proposed mechanisms include catecholamine excess, coronary artery spasm, and microcirculatory dysfunction. Postmortem examinations of hearts that displayed characteristics of stress-induced cardiomyopathy do not show overt pathology in the majority of cases. However, microscopic analysis reveals myofibrillar degeneration, myocytolysis, and inflammatory cell infiltration unevenly distributed

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throughout the heart, but most dense at the apex and ventricular subendocardial areas.

Crisis Management

Treatment of the underlying neurologic insult will aid in the resolution of myocardial stunning. Patients with significant heart failure and hemodynamic compromise will need inotropic and vasopressive support for a period of time. Early involvement of a cardiologist is recommended.

Key Points

- Onset subsequent to an acute emotional or physiologic stress such as brain injury.
- Similar presentation to acute MI, therefore, must differentiate between the two.
- Typical echocardiographic appearance of apical ballooning without angiographic critical lesions.
- Inotropic and vasopressive support may be necessary.
- Complete resolution of the apical wall motion abnormality and depressed cardiac function typically within 48 h after the initial insult and with successful treatment of neurologic crisis.

Cardiac Arrhythmias

Many cardiac arrhythmias can occur in neurologically injured patients with known cardiac disease as well as in those without. Some arrhythmias are attributable to coronary artery insufficiency or ischemia, but others are due to conduction disturbances that result from the neurological illness itself. The most frequent arrhythmias following brain injury are premature ventricular complexes, sinus arrhythmia, and atrial fibrillation. Other arrhythmias including atrial flutter, ventricular tachycardia, torsades de pointes, ventricular fibrillation, and asystole have been documented as well. SAH patients have arrhythmias about 35–85% of the time. Life-threatening arrhythmias occur in <5% of patients with SAH. As with neurogenic myocardial stunning, resolution of the acute intracranial pathology, e.g., normalization of the ICP, injury generally leads to improvement or resolution of arrhythmias.

Supraventricular Tachycardia/Arrhythmia

Overview

Supraventricular tachycardia (SVT) such as sinus tachycardia, atrial fibrillation, and paroxysmal supraventricular tachycardia (PSVT) is common in all critical care settings. Sinus tachycardia (HR >100/min) usually represents a physiologic response to pain, stress, hypotension, heart failure, or excessive catecholamine drive.

Atrial fibrillation (A-fib) with or without a rapid ventricular response (RVR) is commonly seen with acute neurologic insults particularly in the elderly. A fair number of patients manifest atrial arrhythmias, mainly atrial fibrillation, within the first few days after stroke. Occasionally, a cardiac arrhythmia will actually be the initial event that provokes brain injury because of clot formation in the heart that embolizes into the brain (ischemic stroke). However, in other cases, the original injury is in the brain, which then is associated with cardiac rhythm disturbances. RVR is often an urgent issue as it can precipitate a demand ischemia of the myocardium or may compromise cardiac function. PSVT is related to a reentry or similar mechanism at the AV node. See Table 88.1 for management.

Table 88.1 Supraventricular arrhythmia crisis management

Sinus tachycardia	Analgesia and sedation as appropriate, fluid and electrolyte replacement, resolution of the precipitant
Atrial fibrillation	Determine chronicity – if new onset is less than 48 h old, then chemical or electrical cardioversion can be instituted. If more than 48 h since onset or undetermined delay cardioversion until anticoagulation can be instituted. Echocardiography to evaluate for clot formation prior to cardioversion. Beta-adrenergic blockers first line – labetalol or esmolol
	Diltiazem and digoxin, if hypotension is a problem
PSVT	Vagal stimulation maneuvers – carotid sinus massage. Adenosine, amiodarone, diltiazem, and beta-blockade can all be effective

Prevention

Cardiac monitoring for all ICU patients, adequate pain control, assessment of volume status, adequate sedation, careful assessment of electrolyte balance, and treatment of precipitating cause is paramount to prevention (Table 88.1).

Ventricular Arrhythmias

Overview

Ventricular tachycardia, ventricular fibrillation, and torsades de pointes are less common than atrial tachycardias in the neurocritical care unit. These particular arrhythmias are likely a cause for sudden death in patients with a significant neurologic insult. Isolated premature ventricular contractions (PVCs) are common and do not require treatment, but if they occur with increasing frequency, they may signify elevated ICP and the risk for serious ventricular arrhythmias.

Table 88.2 Ventricular arrhythmia crisis management

Ventricular tachycardia (VT)	For significant hemodynamic compromise cardioversion and ACLS guidelines as appropriate
	If hemodynamically stable – amiodarone, then cardioversion
	Polymorphic VT gives magnesium sulfate IV and cardioversion
Ventricular fibrillation (VF)	Early defibrillation according to ACLS guidelines
Torsades de pointes	Stop all QT-prolonging medications, magnesium sulfate IV, and cardioversion

Ventricular flutter and fibrillation are more commonly seen in patients with underlying ischemic heart disease. See Table 88.2 for management.

Prevention

All patients with a neurologic injury or insult need cardiac monitoring until the acute phase of the illness has resolved, as prompt treatment will be necessary for ventricular arrhythmias. Monitoring of QT intervals is important as a prolonged QT places a patient at risk for ventricular ectopy.

Bradycardia

Overview

A HR below 60 bpm is usually the result of sinus node dysfunction or an atrioventricular conduction disturbance. Acute cerebral insults can also produce a vasovagal response. Bradycardia can be seen after carotid angioplasty and stenting procedures from direct and prolonged carotid sinus stimulation as well. Bradycardia in a neurocritical care patient is a red flag for increased intracranial pressure. The triad of bradycardia, hypertension, and respiratory depression is, of course, termed the "Cushing reflex" and results from acutely increased intracranial pressure. Always consider increased ICP as one of the differential diagnoses for bradycardia and hypertension in the context of neurologic injury. This is of particularly concern, if the disease process is suspected in the posterior fossa.

Crisis Management

Cardiac monitoring and careful assessment of a patient's neurologic exam and ICP monitoring as appropriate is paramount to identify the precipitating causes. Consider anticholinergic drugs such as atropine and glycopyrrolate and transcutaneous or transvenous pacing in the presence of significant hemodynamic compromise.

Key Points

- Arrhythmias occur in the neurologically injured with and without intrinsic cardiac disease.
- All brain-injured patients need cardiac monitoring until the acute phase of illness has passed.
- SAH patients have arrhythmias about 35% of the time. Life-threatening arrhythmias occur in <5% of patients with SAH.
- Ventricular arrhythmias are less common than supraventricular arrhythmias.
- For life-threatening arrhythmias institute cardioversion and ACLS early.
- Chronicity of atrial fibrillation should be determined before cardioversion because of the possibility of mural thrombus and the risk for embolic stroke.
- Bradycardia may be a sign of increased ICP as part of Cushing's triad.

Blood Pressure Disturbances in the Neurocritical Care Unit

The central nervous system (CNS) is susceptible to extremes in blood pressure (BP) fluctuations. Hypertension can be associated with increased risk of bleeding and cerebral edema. Hypotension can be associated with infarcts or global ischemia. Cerebral blood flow (CBF) is relatively constant over a wide range of systemic blood pressures in a healthy brain.

The autoregulation curve is shifted rightward in chronically hypertensive patients (CBF remains stable at higher mean arterial blood pressures (MAP) but becomes flow passive that is prone to ischemia already at low-normal MAP). This section focuses on blood pressure management in the setting of subarachnoid hemorrhage, intracranial hemorrhage, and ischemic stroke.

Hypertension in SAH

Overview

Blood pressure management in SAH differs according to the presence of an aneurysm, if that aneurysm has been surgically secured, and the presence of additional/residual aneurysms. Hypertension is common immediately following aneurysmal rupture and often reflecting a hyperadrenergic state and/or increased ICP with a Cushing's response. For hypertension in ruptured and unsecured aneurysms, maintaining systolic BPs in a range for adequate perfusion while avoiding rapid and extreme changes in BP is paramount to avoid shear stress on an aneurysm. Shear stress places the patient at risk for rebleeding. Generally systolic BP goals should be between 120 and 160 mmHg while keeping a cerebral perfusion pressure (CPP)

>70 mmHg to avoid exacerbation of cerebral ischemia. CPP is calculated as MAP – ICP or CVP, whichever is greater – and it ranges normally between 70 and 100 mmHg. After an aneurysm has been secured, BP goals should shift in an upward direction due to lessened risk of bleeding and the increased risk of vasospasm. Permissive hypertension is a strategy after an aneurysm is secured to obtain higher perfusion pressures with the goal to prevent further brain ischemia. Arterial hypertension is frequently even induced therapeutically for the treatment of cerebral vasospasm. The technique involves the use of vasopressors and IV fluids to achieve a higher than normal MAP and thus augment the CPP. These therapies have the goal of preventing or ameliorating brain ischemia, which is created by cerebral vasospasm.

Prevention

All patients with SAH should have beat to beat blood pressure monitoring via an arterial line in addition to cardiac monitoring. Initial prevention of hypertension in SAH includes initiation of sedation, analgesia, antiepileptic therapy, and nimodipine (a calcium channel blocker shown to improve outcome in SAH) all of which lower blood pressure.

Crisis Management

Blood pressure management (see Table 88.3) is used in conjunction with ICP control as appropriate (hyperventilation, head of bed elevation >30°, anti-edema therapies, CSF drainage), as well as surgical evaluation for clipping and coiling of aneurysms as determined by a neurosurgeon to prevent further injury.

Hypertension in Intracerebral Hemorrhage

Overview

Intracerebral hemorrhage (ICH) includes nontraumatic brain injuries with bleeding into the epidural, subdural, subarachnoid, intraventricular, and intraparenchymal spaces. There are many causes for nontraumatic ICH. Hypertension is an important cause for ICH, and it is of utmost importance to control for the prevention of further bleeding leading to hematoma expansion and poor outcomes. Management of BP is individualized depending on the cause of the ICH. Patient factors such as chronic hypertension (which shifts the CBF autoregulation curve to the right), age, and time from the hemorrhage are all important factors to consider.

Table 88.3 Therapeutic hypertension goals in SAH with vasospasm

Secured aneurysm	Titrate vasopressors to SBP 180–200, DBP 100–120, MAP 120–140
Unsecured aneurysm	Titrate vasopressors to SBP 160–170, DBP 90–100, MAP 100–120

Table 88.4 Commonly used antihypertensive medications

Drug	Dose
Labetalol	5–10 mg IV q10′ as needed
Enalaprilat	
Hydralazine	0.625–1.250 mg IV q6h as needed
Esmolol	2.5–10 mg (up to 40 mg/dose) IV q4–6 h as needed
Nicardipine	0.25–0.5 mcg/kg load; 50–200 μg/kg/min

Prevention

All patients, who are at risk for rebleeding after ICH, should remain under intensive care surveillance and have beat to beat blood pressure monitoring via an arterial line in addition to cardiac monitoring. Initiation of analgesia and sedation (with close monitoring of neurologic function) as appropriate are initial steps for blood pressure management.

According to the American Heart Association (AHA) guidelines treat for:

- SBP >230 mmHg or DBP >140 × 2 readings 5 min apart – sodium nitroprusside
- SBP 180–230 mmHg or DBP 105–140 mmHg or MAP >130 mmHg × 2 readings 20 min apart – IV labetalol, esmolol, enalaprilat, or nicardipine

Crisis Management

Goal CPP with ICP monitoring is >70 mmHg.

Blood pressure management is used in conjunction with ICP control (hyperventilation, head of bed elevation >30°, anti-edema therapies) as well as surgical evacuation to prevent further injury. See Table 88.4 for suggestions of antihypertensive medications.

Hypotension in ICH

Overview

Determination of hypotension depends on the patient's history and clinical picture, the presence of elevated ICP, and the cause of an ICH. Hypotension is relatively uncommon in ICH.

Prevention

Close monitoring of BP and assessment of a patient's volume status are paramount to the prevention of hypotension, which can place a patient at risk for or exacerbation of brain ischemia.

Crisis Management

Treatment with volume replacement is usually first line avoiding hypotonic and glucose-containing solutions. Normal saline and hypertonic saline are the fluids of choice as appropriate. Vasopressors may be indicated.

Hypertension in Ischemic Stroke

Overview

Most ischemic stroke patients become hypertensive after the onset of symptoms. In addition, many affected patients are hypertensive at baseline as one of the risk factors for the disease. Hypertension during the acute poststroke time period is usually thought of as beneficial, to improve perfusion to the ischemic penumbra via collateral circulation. Balancing the potential benefits of hypertension with the risk of hemorrhagic transformation of a large stroke is an important consideration.

Prevention

All patients with ischemic stroke, who are critically ill, should have beat to beat blood pressure monitoring via an arterial line in addition to cardiac monitoring. Consider careful sedation and analgesia, if appropriate with close monitoring of neurologic function.

Crisis Management

The specific blood pressure targets established by the American Heart Association depend on whether the patient has received IV tissue plasminogen activator (tPA). The rationale for this is that patients who have received IV tPA are at higher risk of bleeding complications. For patients after ischemic stroke who have not received tPA, permissive hypertension should be allowed to a SBP up to 220 mm Hg. For patients who have received tPA or thrombectomy, SBP should be lowered only when it rises above SBP 180 mm Hg. Any patient who suffers a hemorrhagic transformation of their stroke should have blood pressure maintained below SBP 140 mm Hg.

Key Points

- Hypertension is common following SAH, ICH, and ischemic stroke and is secondary to the underlying pathophysiology, a hyperadrenergic state, and increased ICP and need for elevated CPPs.
- Blood pressure management is crucial in SAH to prevent further ischemic damage secondary to rebleeding, increased ICP, and vasospasm.
- Hypertension is an important cause for ICH blood pressure control which is paramount to prevent hematoma expansion and poorer outcomes.
- Treatment of hypertension in ischemic stroke should be avoided unless BP is profoundly elevated SBP >220 mmHg or MAP >130 mmHg, or IV thrombolytics were given (tight control for 24 h).

Suggested Reading

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