Embolic Stroke

Prior to making any medical decisions, please view our disclaimer.

Clinico-radiographic Embolic Syndrome

Features suggestive of embolization may include:

1. Sudden onset of symptoms, with severity maximal at onset
2. Cortical or subcortical lesion localization (by symptoms or imaging) to the territory supplied by the major branches of the ACA, MCA or PCA in the absence of proximal vascular stenosis/occlusion
3. Clinical or radiographic evidence of embolic infarcts to multiple vascular territories (CNS or systemic), or to the grey-white matter junction
4. In the lenticulostriate or thalamogeniculate territories, lesions greater than 1 cm should raise suspicion of embolism
5. Cerebellar hemispheric infarction
6. The identification of a possible embolic source in the heart (e.g. left atrial or ventricular thrombus, atrial myxoma, atrial fibrillation, valvular disease, cardiomyopathy with ejection fraction < 25%, or acute myocardial infarction), aortic arch or great vessels (thrombus or cholesterol, arterial dissection), venous system in association with intracardiac shunt (fat, tumor, air, deep vein thrombus) or systemic circulation (hypercoagulability, hyperviscosity. The failure to identify a high risk source of embolism (cryptogenic, up to 40% of all ischemic strokes) does not exclude an embolic mechanism. Other risk factors for stroke have been suggested by large clinical trials or observational data, including calcified posterior mitral annulus, PFO with or without septal aneurysm, left atrial spontaneous echo contrast, valve strands, atrial flutter, cardiomyopathy with ejection fractions > 25%.
7. Peri-procedure event in setting of known risk of embolism (cardiac or great vessel surgery, angiography or endovascular manipulation)

Phase 1

Additional Diagnostic Testing

- Consider transthoracic echocardiography with agitated saline
- Consider transesophageal echocardiography with agitated saline to exclude aortic arch atheroma, mitral valve vegetation, and to better image the left atrium and atrial appendage. TEE may also help characterize PFO/ASD when considering closure
- Consider hypercoagulable labs
- Consider 24-hour holter monitor
- Consider possibility of infective and marantic endocarditis. Avoid heparin if strongly suspected
- Rule out MI
- If evidence of PFO, rule out venous thrombus with LENI's and pelvic MRV
- If artery-to-artery embolism is suspected, see management algorithm in Large Vessel section.

Prevention of Acute Recurrent Stroke

- Assess risk of re-embolization during course of acute hospitalization and consider anticoagulation with unfractionated heparin, LMW heparin or heparinoids unless contraindications for anticoagulation exist (e.g., large area of infarction at significant risk of hemorrhagic transformation, infective endocarditis). Avoid heparin bolus unless suspected basilar embolism or at high risk for re-embolism or clot propagation.
- Use hirudin or other non-heparin anticoagulants in patients with HIT.
- Consider induced hypertension to determine if symptoms can be ameliorated with augmented CBF. Patients with patent circle of Willis by CTA or MRA may not benefit from induced hypertension and may be at greater risk of ICH.
- Consider neuroprotective therapies
- Consider neurosurgical consultation in cerebellar infarction at risk for brainstem compression or hydrocephalus.

Phase 2

Subacute Medical Management

Communicate with PCP
• Special considerations based on lesion location and pathophysiology (see link below)
• Avoid acute BP reductions except in setting of coronary ischemia or impaired cardiac contractility and/or signs of end-organ failure
• Maintain euthermia, euglycemia, eunatremia
• Consider Intensive Care management (e.g., airway compromise, severe hypertension, acute MI, cerebral edema, hydrocephalus, major organ dysfunction)
• Consider hemicraniectomy for hemisphere refractory cerebral edema and impending herniation
• Repeat imaging to assess stroke evolution and hemorrhagic transformation

Ongoing Assessments:

• DVT risk if immobile
• Ability to urinate; UTI
• Ability to swallow; Aspiration; need for feeding tube
• GI/GU bleeding
• Cardiopulmonary function; need for tracheostomy

Functional Assessment and Acute Rehabilitation

Communicate with other healthcare personnel

• safety for ADL, ambulation
• urinary and fecal continence
• Tone and splinting
• Exercise tolerance
• Cognitive function
• bedside speech and swallow evaluation

Phase 3

Discharge Planning

Discuss with patient and family:

1. treatment and prognosis
2. risk factors and risk reduction strategies

Assess subacute rehabilitation needs and eligibility (consider PM&R consult)

1. Short v. long term care needs
2. Inpatient rehabilitation v. skilled nursing facility rehabilitation
3. Home v. outpatient services

Assess financial resources to cover cost of:

1. Inpatient, outpatient rehabilitation services
2. Medications
3. Assistive devices (e.g., commode, cane, wheelchair, hospital bed, etc.)
4. Long term care

Long term Secondary Prevention

Risk factor modification

1. Hypertension control
2. Lipid reduction
3. Smoking cessation
4. Nutrition counseling and glycemic control
5. Weight reduction and increased physical activity
6. Anticoagulation or Antiplatelet therapy
Medical Considerations

1. Duration of anticoagulation +/- antiplatelet agent
2. Consider PFO closure
3. Consider statins for mild hyperlipidemia
4. Provide adequate followup with stroke specialist
5. Consider secondary prevention or recovery trials

Patient education

1. Patient/family understands stroke diagnosis
2. Patient/family understands how to lower risk

Authoring Information

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Last reviewed: 4/8/2010

Last updated: 4/8/2010