SCREENING FOR BLUNT CEREBROVASCULAR INJURIES (BCVI)

Goal

- identify patients with a high probability of injuries to the carotid and vertebral arteries in blunt trauma
- reduce the incidence of neurologic sequelae
- early identification of at high risk patients who might benefit from early screening imaging

Guidelines:

I. <u>Screening criteria (i.e. asymptomatic patients)</u>

- High-energy transfer (e.g. MVC, significant fall) with:
    - Leforte II or III fracture
    - Complex mandibular fracture (isolated mandibular fracture with a low-energy mechanism does not require screening – e.g. punch to the jaw)
- All cervical-spine fractures except isolated spinous process fractures
- Severe cervical hyperextension/rotation or hyperflexion
- Basilar skull fracture with carotid canal involvement
- Diffuse axonal injury with Glasgow Coma Scale (GCS) < 6
- Near-hanging injuries

II. <u>Indication for prompt evaluation to rule out BCVI</u>

- Arterial hemorrhage from mouth, ears, nose and neck
- Cervical bruit in patient <50 years of age
- Expanding cervical hematoma
- Unexplained focal neurological deficit or neurological exam incongruous with findings on head CT scan
- Evidence of ischemic stroke on CT
- Chest trauma

Method and timing of screening

Patients meeting screening criteria should be evaluated by CT angiography (CTA), specifically requesting the 4 vessel CTA protocol. Patients should be screened within 24 hrs of injury unless extenuating circumstances preclude imaging (hemodynamic instability, difficulty with ventilation, concern of contrast-induced nephropathy, etc). Preference is given to perform the CTA at the first round of CT scans at admission. The benefits of antiplatelet/anticoagulant therapy are typically outweighed by the risk of bleeding within the first 24 hrs.

Further evaluation and treatment

Patients with an identified injury on CT angiography require assessment by the neurology service for further evaluation and management. There is accumulating evidence suggesting that early identification of BCVI with a therapeutic plan based on the location and severity of injury might reduce the incidence of neurologic sequelae. Intervention might take the form of antiplatelet agents, anticoagulation or surgical/angiographic intervention depending on the nature of the injury and clinical setting. Early identification prior to the development of neurologic deficit requires a method for screening patients at highest risk.