Identification and Management of Vertebral Artery Injury Associated with Cervical Trauma

Peter G. Whang, MD, FACS
Associate Professor, Spine Service
Department of Orthopaedics and Rehabilitation
Yale University School of Medicine

Anatomy
- Vertebral artery (VA) arises from subclavian artery
- 4 segments
  o V1 – extraosseous – proximal to transverse foramen
  o V2 – foraminal – within trasverse foramina from C6 to C1
  o V3 – extraspinal – distal to C1 transverse foramen along arch
  o V4 – extradural – unite to form basilar artery
- 10% of individuals exhibit unilateral VA hypoplasia

Types of VA injuries (VAI) – Denver Radiologic Scale
- Intimal tears – flap within lumen (Grade I)
- Dissections – false lumen within wall (subintimal vs. subadventitial, Grade II)
- Pseudoaneurysms – rupture through wall with extravascular hematoma (Grade III)
- Occlusion (Grade IV)
- Transection – usually fatal (Grade V)

Incidence
- Blunt VAI are uncommon and reported rates vary according to method of diagnosis
- Detection increasing with greater index of suspicion, prevalence of screening, and advancements in diagnostic modalities
- Observed in approximately 0.5% of all patients with blunt trauma
- Bilateral involvement in up to 28% of VIA cases
- Clearly association with cervical fractures (up to 70%)
- Most likely to occur within V2 segment

Mechanism of Injury
- Direct trauma from bony fracture fragments
- Excessive traction/shear forces
- Associated with distraction/extension, distraction/flexion, and lateral flexion forces
- Most frequently occur as a result of high energy trauma (MVA, fall, sports injury, assault, hanging)

Pathophysiology
- Majority of unilateral VAI are benign (rich collateral circulation, recanalization)
- Thrombosis +/- embolism may give rise to TIA/ basilar stroke, spinal cord infarction, or even death (4-8% mortality rate)

Clinical Presentation
- Related to disruption of posterior circulation
  - Dysarthria
  - Gait imbalance/impaired coordination
  - Visual field deficits/diplopia
  - Horner’s syndrome
  - Lower cranial nerve palsies
  - Impaired consciousness
- Signs/symptoms usually present within 72 hours of injury but sequelae may occur in delayed fashion (vasospasm)

Risk Factors
- Fractures involving transverse foramen
- Subluxation/dislocation
- Fractures involving proximal cervical spine (C1-C3)

Diagnosis
- Catheter cerebral angiography remains gold standard but associated with risk of iatrogenic injury/stroke
- CT angiography – may be preferred method
- MR angiography
- Doppler ultrasound less accurate because of surrounding bony structures
Screening
- Widely implemented because findings may have significant implications on treatment and follow-up
- Because incidence of VAI is relatively low and most patients are asymptomatic, screening criteria remain a matter of debate – based on mechanism of injury, clinical/radiographic features
- Denver criteria
  - Cervical fracture/dislocation
  - Neurologic deficits not explained by cerebral imaging
  - Horner’s syndrome
  - Facial fractures (LeFort II/III)
  - Skull base fracture

Treatment
- Optimal management strategies for VAI has not yet been established
- Paucity of Level 1 evidence from prospective RCT available to guide practitioners
- Treatment recommendations vary according to type of VAI
- Wide range of treatments have been advocated for symptomatic lesions
  - Anticoagulation/thrombolytic therapy
  - Endovascular procedures – thrombolysis with stent placement, embolization
  - Surgery – rarely performed because of high risk of morbidity/mortality (uncontrolled hemorrhage)
- Asymptomatic patients may simply be observed but prophylactic anticoagulation often administered when not contraindicated but risk reduction attributable to these regimens has not been elucidated
- Timing of surgical fixation of concomitant cervical injuries is still controversial
Bibliography