Posterior Circulation Stroke in Children Due to Anomalies of the Cervical Spine

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Introduction

Vertebral artery dissection due to dynamic compression by abnormal C1-C2 anatomy is an under-recognized cause of childhood stroke. Our goal was to describe pathologic variants of soft tissue or bone structures at the C1-C2 level in children who presented with vertebral artery dissection and posterior circulation strokes.

Methods

A series of five children diagnosed with vertebral artery dissection from compression by C1-C2 soft tissue or bone were identified at a single pediatric stroke center. Diagnostic evaluation, anatomic cause of dissection, and treatment were abstracted from medical records.

Thromboembolism from Vertebral Artery

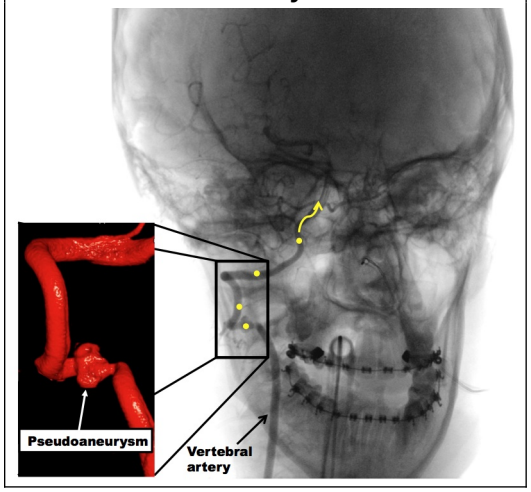

Figure 1. Vertebral artery pseudoaneurysm leading to thrombus formation and embolus.

Angiographic Findings in Two Patients

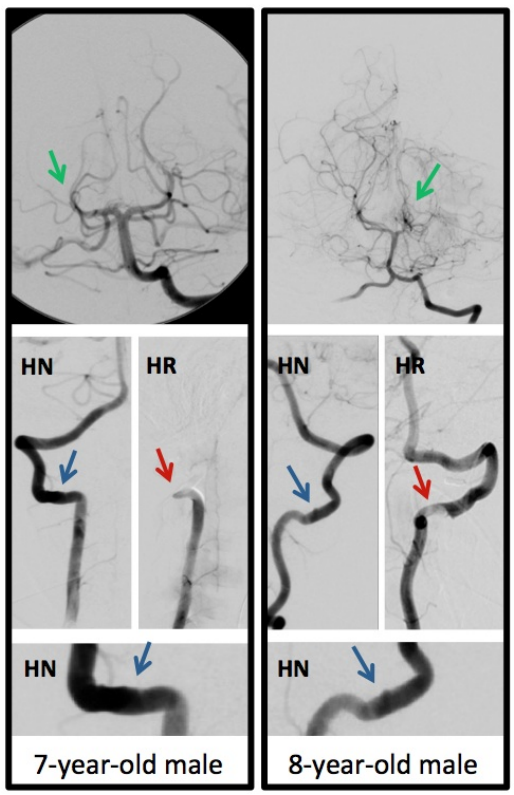

Figure 2. HN – Head in neutral position. HR – Head rotated (contralateral >70 degrees). Posterior circulation angiograms show branch occlusions (green arrows); subtle focal irregular and/or fusiform dilation (blue arrows) in the proximal V3 loop of the vertebral artery reflecting vertebral artery dissections (VAD) of varying chronicity; at times small intraluminal thrombus is present at the site of VAD (not shown). With head rotation 70 degrees contralateral to the injected side, extrinsic compression causes focal occlusion at the same location (red arrows).

Results

Five children (all boys, ages 6 - 14) were identified with a diagnosis of posterior circulation embolic strokes due to focal vertebral artery dissection and presumed compression against adjacent C1-C2 structures. Four had recurrent events, including basilar artery thrombosis, before the cervical abnormality was identified. Vertebral artery dissections were caused by bony (congenital arcuate foramen, post-traumatic os odontoideum) and soft tissue pathology (ruptured atlantoaxial bursa, anomalous C1-C2 connective tissue bands). Vertebral artery compression at dissection sites during head rotation was confirmed by angiography with head in neutral and rotated in children with soft tissue anomalies. No other etiology for stroke or dissection was found in any of the cases. For secondary stroke prevention, four children underwent direct surgical decompression of the vertebral artery or spinal fusion; one opted for activity restriction and aspirin. None had further recurrences after treatment with a median follow-up of 18 months (range 1 to 61).

Conclusions

In children with recurrent vertebral artery dissection and strokes, C1-C2 bone imaging and dynamic angiogram with neck rotation can identify cervical pathology that may otherwise be missed. Cervical stabilization or removal of the compressive pathology should be considered to prevent stroke recurrence.

Learning Objectives

By the conclusion of this session, participants should be able to: 1) Describe the significance of spontaneous posterior circulation strokes in children, 2) Discuss how cervical spine anomalies can result in vascular injury, 3) List effective treatments to prevent strokes caused by vertebral artery injury.

Vertebral Artery Decompression

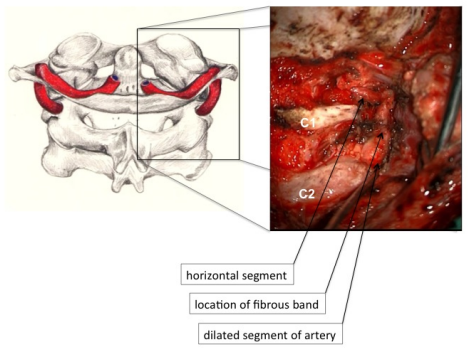

Figure 3. Vertebral artery decompression with removal of bony and soft tissue structures.

References

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