The Diagnostic Role of MR Intracranial Wall Imaging?

ASA/ISC Symposium
Nonatherosclerotic Intracranial Arterial Stenoses
Thursday Feb 2\textsuperscript{nd}, 2012

Richard H. Swartz
rick.swartz@sunnybrook.ca
Acknowledgements

**UHN Neuroradiology and Imaging**
Drs. D. Mikulis, D. Mandell
Farb, Krings, Agid, terBrugge, Willinsky, S. Bhuta, D. Johnstone

**University of Toronto Stroke Program**
Drs. Black, Casaubon, DelCampo, Gladstone, Hopyan, Jaigobin, Saposnik, Selchen, Silver, Vergowen, Ween; J. Breaton.

**Sunnybrook Neuroradiology and Imaging**
Drs. Aviv, Fox, Howard, Symons, Yeung
Drs. Graham and MacIntosh

**Sunnybrook Stroke Program**
**Sunnybrook Stroke Research Unit**
“Beyond the lumen”

- Conventional “lumenology” can lack specificity – different pathologies can cause similar narrowing in blood flow
- Imaging of coronary and extracranial carotid arteries going “beyond the lumen” – how does this apply to intracranial vessels?
Intracranial vessels are unique

- Inaccessible for pathology – intracranial vasculopathies can only be imaged
- Requires high spatial resolution
- Multiple pathologies to differentiate
  - Atherosclerotic (one of the most common causes of stroke worldwide, with high recurrence risk)
  - Vasculitis, Reversible Cerebral Vasoconstriction syndrome, Moya-moya, ?chronic focal inflammatory vasculopathy
Intracranial Wall Imaging

• Centered on intracranial vessel of interest

• Using 3-T MRI (GE Signa)
  • 3 mm axial T2 FRFSE
  • 3 mm T1 FLAIR pre-contrast; axial, coronal +/- sagittal (6.6 mins each)
  • 3 mm T1 FLAIR post-contrast; axial, coronal, +/- sagittal

• T1 FLAIR is a “black blood” sequence

Intracranial arterial wall imaging using high-resolution 3-tesla contrast-enhanced MRI

Neurology® 2009;72:627-634
Features of the vessel wall on MR

• Wall thickening (T2; T1 FLAIR pre-contrast)
  • Eccentric (can be circumferential; smallest side <50% largest)
  • Concentric

• Pattern of Gadolinium enhancement
  • Presence of enhancement (?vasa vasorum, ?inflammation, ?hemorrhage)
  • Degree of enhancement
Wall thickening hypotheses

- **Atherosclerosis**
  - Eccentric irregular wall thickening

- **Inflammatory disease**
  (Vasculitis and RCVS)
  - Smooth circumferential concentric wall thickening

- **Dissection**
  - Eccentric wall thickening with T1 bright hematoma +/- flap

- **Moya-moya**
  - At diagnosis (late): atrophy, stenosis, no thickening

*1st 3 blood vessel art courtesy of Dr. D. Mikulis*
Wall thickening: Atherosclerosis (focal, eccentric)
Focal eccentric thickening of the basilar artery wall

R.H. Swartz, University of Toronto
MCA atherosclerosis

- Patients with \( \geq 2 \) atherosclerotic risks, MCA stenosis, recent symptomatic stroke
- N=8 patients.
  - 6 had eccentric M1 stenosis
  - 1 eccentric proximal M2 stenosis
  - 1 distal M2 stenosis – inconclusive eccentricity
- All recently symptomatic; all enhanced
MCA atheroma – eccentric & enhancing
Vasculitis

- 67 y.o. F multiple TIA’s, followed by headache and multifocal neurological deficits rapidly progressed to decreased level of consciousness.
- DWI showed dozens of small cortical and subcortical areas of restriction. CTA showed bilateral narrowing of cavernous and supraclinoid ICA’s.
- Temporal artery biopsy: destruction of internal elastic lamina, multi-nucleated giant cells.

R.H. Swartz, University of Toronto
Vasculitis

Biopsy proven CGA with smooth, diffuse, concentric intra-cranial wall enhancement.
Reversible Cerebral Vasoconstriction

- Sudden, severe HA, multifocal cerebral arterial narrowing, absence of aneurysmal subarachnoid blood, near-normal CSF

- For most definitions, differentiation from CNS vasculitis requires resolution within 3 months.

- RCVS affects regulation of arterial tone, limited pathology suggests lack of inflammation…
Reversible Cerebral Vasoconstriction: Concentric wall thickening. NO enhancement (n=3). Vessel resolved.

A. Pre-contrast

B. Post-Gadolinium contrast

C. At presentation

D. At follow-up

Mandell et al, Stroke online Dec 2011
Vasculitis: concentric wall thickening, enhancement (n=4). Persistent vascular anomalies.
Moya-moya

• Initial series (Neurology, 2009) reported 2 cases with Moya-moya syndrome
• One presented with hemorrhage, the other with hemodynamic ischemic symptoms
• Bilateral severe or occluded MCA M1’s with extensive collateralization
• No wall thickening of ICA’s or MCA’s
• No enhancement (including distal ICA)
Dissection

- 60 year old man with neck pain after a rugby injury (!)
- Presented with ataxia, left sided weakness.
- Basilar artery thrombus, right pontine stroke and left thalamic and temporal artery stroke
**Summary of wall imaging findings**

“There are more things in heaven and earth, Horatio, Than are dreamt of in your philosophy”.

W. Shakespeare, Hamlet; Act 1, scene V

<table>
<thead>
<tr>
<th>Disease</th>
<th>Thickening pattern</th>
<th>Enhancement pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis</td>
<td>Eccentric</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(if symptomatic)</td>
</tr>
<tr>
<td>Vasculitis</td>
<td>Concentric</td>
<td>+</td>
</tr>
<tr>
<td>RCVS</td>
<td>Concentric</td>
<td>-</td>
</tr>
<tr>
<td>Moya-Moya</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Dissection</td>
<td>Irregular +/- flap</td>
<td>T1 bright +/- enhancement</td>
</tr>
</tbody>
</table>
How can we use it?

- Mostly studied “clinically definite” populations (no other gold standard).

- Don’t need 60+ mins at 3T to know what process is affecting an 85 y.o. male smoker with HTN, DM2.

- What about clinically “indefinite”? Children? Isolated stenoses? Young people without any risk factors? People with multiple possible etiologies?

- 53 year old female, PMH: hypertension, high cholesterol, obese, strong family history, on OCP.

- Also: prior miscarriages, ANA strongly ++; RF +
...on antiplatelet, statin, BP treatment. Steroids added.
What can wall imaging do?

- 3T intracranial vessel wall imaging may be able to:
  - Visualize wall of medium intracranial Circle of Willis vessels.
  - Identify atheromatous plaque (eccentric thickening), inflammatory diseases (concentric thickening: vasculitis with enhancement; RCVS without), moyamoya and dissection.
  - Identify components of large atheromatous plaques (lipid core, fibrous cap).
  - ? New pathologies of intracranial circulation ? Focal inflammatory conditions – acute vs. chronic?
Challenges

• Lack of histopathological gold standard
• Variability (vendor, version, coil, sequence…)
• Long scan times (goal: isotropic)
• Likely overly simplistic categorical framework
• Need to solve technical challenges to facilitate broader uptake
• Need broader uptake and broad collaboration to evaluate real-world utility
• Need extensive experience with clinical “gold standard” cases to be confident in diagnoses of complex/unusual/new phenomena.
Opportunity

- Collaborative efforts to ensure comparability across sites, vendors, versions and sequences
- Standardize definitions
- Identify new conditions
- Observe evolution of old conditions
- Improve selection of patients for interventional or empiric medical therapies
- Potential biomarkers against which new treatments for common, severe intracranial pathology can be tested.
Thank-you

UHN Neuroradiology and Imaging
Drs. D. Mikulis, D. Mandell
Farb, Krings, Agid, terBrugge, Willinsky, S. Bhuta, D. Johnstone

University of Toronto Stroke Program
Drs. Black, Casaubon, DelCampo, Gladstone, Hopyan, Jaigobin, Saposnik, Selchen, Silver, Ween; J. Breaton.

Sunnybrook Neuroradiology and Imaging
Drs. Aviv, Fox, Howard, Symons, Yeung
Drs. Graham and MacIntosh

Sunnybrook Stroke Program
Sunnybrook Stroke Research Unit