**Transcranial Doppler Ultrasonography Detection of Small Vessel Knock in Patients With MRI-negative Stroke-like Deficits**

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Transcranial Doppler Ultrasonography (TCD) has been used to detect large vessel occlusion in stroke. Here I report a new TCD finding of small vessel knock (SVK) in 6 cases of MRI-negative stroke-like deficits. MRI-negative stroke-like deficits have been reported before and are thought to be caused by small vessel occlusion without cytotoxic oedema. The small vessel knock (SVK) found in these cases resembles the knock found in circulatory arrest due to cerebral death (Figure 1) and that found in association with large vessel occlusion (Figure 2). SVK has a characteristic appearance and can be detected in association with MRI-positive small vessel occlusive stroke (Figure 3). Figure 4 shows that the SVK is due to occlusion of a small perforating artery. As shown, continuous insonation of the SVK signal can result in opening of the artery with clinical improvement. SVK is not found in normal people, nor in association with the white matter changes resulting from both multiple sclerosis or sporadic CJD (results not shown). Thus, SVK would appear to be specific for small vessel occlusive disease (SVD). The 6 cases presented here all developed sudden onset of stroke-like symptoms with clinical features consistent with the lateral medullary syndrome (LMS) and were SVK-positive but CT and MRI-negative.

**Methods**

A 2 MHz probe was used for insonation via the transforaminal window (SonoSite, USA) with no filter. Diagnostic power settings were used. SVK is found in the 300 Hz region of the spectra which is normally “filtered-out” in most TCD machines. It has a characteristic appearance of a white high intensity signal seen at maximum systole with a reflected wave in diastole (not always visible) (see Figures 3, 4 and 7). SVK is difficult to visualize because it is often overlapped by a much larger arterial signal coming from the main supplying artery (Figure 5). The knock also becomes more obvious during insonation.

**Cases**

The mean age of the 6 cases was 42 (SD 10.6) with a range 27-58. Neurological symptoms in all cases were sudden in onset. Case 1 was a 27 year old male who awoke with right-sided occipital headache, vertigo, diplopia mainly to the right, paraesthesia on the left arm and chest, and left side of face with loss of the corneal sensation on the left. He had been given tPA for a suspected occlusive syndrome due to ischaemia of the lowest medulla or upper cord due to a vertebral artery dissection. He had been given tPA for a suspected myocardial infarction but subsequent cardiac tests have been negative. He is awaiting a coronary angiogram.

Case 2 was a 36 year old female who presented with atypical chest pain, vertigo, diplopia to the right with reduced sensation to pin prick but not touch on the left arm and chest. These symptoms gradually improved over the next few days. This patient had several similar episodes over the next two years. He had no vascular risk factors but had a past medical history of a serious whiplash injury.

Case 3 was a 38 year old male with a past medical history of irritable bowel syndrome who developed sudden onset of chest discomfort with left arm weakness, diplopia to the right and reduced sensation to pin prick on the left arm, chest and right side of face. All subsequent cardiological investigations were negative.

Case 4 was a 45 year old male who had left occipital headache and neck pain for 2 weeks with memory problems and tiredness who then developed sudden onset of loss of vision and chest tightness followed by vertigo, double vision, dizziness and balance problems. He had diplopia to the left > right, with ataxia on the left. He had mild weakness of the left arm and leg with reduced sensation to pin prick only on the right arm and chest and left side of face with loss of the corneal sensation on the left. He had been given tPA for a suspected myocardial infarction but subsequent cardiac tests have been negative. He is awaiting a coronary angiogram.

Case 5 was a 46 year old male diabetic who developed sudden onset of right-sided occipital headache, with diplopia to the right, balance problems and crossed paraesthesia to pain. He also had mild weakness and ataxia on the right.

Case 6 was a 38 year old male who developed sudden onset of right sided occipital headache with, paraesthesia to pain on the left arm, chest and right side of face with diplopia to the right. He also had weakness to the right. He had a reduced flow void in the right vertebral artery on MRI.

**Results**

These cases emphasise the complexity of posterior circulation stroke. All cases had stroke-like episodes with features of the lateral medullary syndrome (see Figure 6). However, Cases 3-6 also had motor weakness, which is not a typical feature of LMS suggesting that more than one territory of the brain stem was involved. Case 3 had crossed weakness and 4, 5 and 6 ipsilateral weakness. In all cases SVK was found in more than one territory of the brain stem was involved. Case 3 had crossed weakness and 4, 5 and 6 ipsilateral weakness. In all cases VA SVK was found in the basilar artery in Cases 4 and 6. The ipsilateral motor weakness found in Cases 4, 5 and 6 could be due to involvement of the posterior cerebral artery (PCA). However, no image is obtained with TCD and so I cannot be certain whether SVK was directly associated with occlusion of a small perforating artery. As shown, continuous insonation of the SVK signal can result in opening of the artery with clinical improvement. SVK is not found in normal people, nor in association with the white matter changes resulting from both multiple sclerosis or sporadic CJD (results not shown). Thus, SVK would appear to be specific for small vessel occlusive disease (SVD). The 6 cases presented here all developed sudden onset of stroke-like symptoms with clinical features consistent with the lateral medullary syndrome (LMS) and were SVK-positive but CT and MRI-negative.

**Conclusions**

Small vessel knock is a new TCD finding which would appear to be both specific and sensitive for small vessel occlusive disease. It can be found in stroke-like syndromes in both MRI-positive and negative cases. Detection of knock involves a new TCD insonation technique (Patent pending), which is easily operator-dependent but can be taught. Insonation of the SVK can result in clinical recovery over a considerable time window. In the above 6 cases of posterior circulation stroke-like episodes with LMS features, SVK was found in more than one location in 4 of these cases. The finding of small vessel occlusion in other areas of the brain stem than the lateral medullary fault consistent with their clinical presentation. It is also consistent with the vertical arrangement of the posterior circulation arteries and explains the preponderance of multiple infarcts at different levels in the posterior circulation. The detection and insonation of SVK signals allows the exciting possibility of targeting small vessel occlusive disease before an MRI becomes positive. This could allow the early detection and treatment of small vessel occlusion in vascular Alzheimers. A randomised control trial of this new technique is planned.

**Table 1**

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Sex</th>
<th>VA depth (mm)</th>
<th>Vascular risk</th>
<th>MRI</th>
<th>MRA</th>
<th>CT scan</th>
<th>VA flow</th>
<th>SVK detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>27</td>
<td>M</td>
<td>RVA 63</td>
<td>Neck injury</td>
<td>normal</td>
<td>normal</td>
<td>RVA &lt; LVA</td>
<td>RVA &lt; LVA</td>
</tr>
<tr>
<td>Case 2</td>
<td>36</td>
<td>F</td>
<td>RVA 67</td>
<td>Neck injury</td>
<td>normal</td>
<td>normal</td>
<td>RVA &gt; LVA</td>
<td>RVA &gt; LVA</td>
</tr>
<tr>
<td>Case 3</td>
<td>38</td>
<td>M</td>
<td>RVA 50, 77</td>
<td>SVH</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>RVA &gt; LVA</td>
</tr>
<tr>
<td>Case 4</td>
<td>45</td>
<td>M</td>
<td>RVA SS, LVA, Bas 78, 91</td>
<td>SH</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>RVA &gt; LVA</td>
</tr>
<tr>
<td>Case 5</td>
<td>46</td>
<td>M</td>
<td>RVA SS, LVA, Bas 78, 91</td>
<td>Diabetic</td>
<td>normal</td>
<td>normal</td>
<td>normal</td>
<td>RVA &gt; LVA</td>
</tr>
<tr>
<td>Case 6</td>
<td>58</td>
<td>M</td>
<td>RVA 55, 72, Bas 86</td>
<td>Smoker, alcohol</td>
<td>RVA reduced</td>
<td>flow void</td>
<td>normal</td>
<td>RVA &lt; &lt; LVA</td>
</tr>
</tbody>
</table>

PM = smoker, PM = PMI, RVA = Right vertebral artery LVA = Left vertebral artery Bas = Basilar artery MRI = Magnetic resonance imaging T1, T2 weighted MRA = Magnetic resonance angiography. VA depth through the transforaminal window-numbers indicate depth of detection of small vessel knock. RVA flow measured in the VA segment of the VA at the base of skull.

**References**

3. Opalski syndrome due to ischaemia of the lowest medulla or upper cord due to occlusion of the posterior inferior cerebellar artery (PICA). However, no image is obtained with TCD and so I cannot be certain whether SVK was directly associated with occlusion of a small perforating artery.