Author's response to reviews

Title: Unusual MRI findings of dural arteriovenous fistula: Isolated perfusion lesions mimicking TIA

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Author's response to reviews: see over
Point-by-point responses to the reviewer’s comments on the manuscript

Title: Unusual MRI findings of dural arteriovenous fistula: Isolated perfusion lesions mimicking TIA

To BMC Neurology Editor,

The authors would like to thank Editor and Reviewers for careful review of our manuscript and providing us with their comments and suggestions to improve the quality of the manuscript.

We reformulated the manuscript with an effort to focus in the main original information on the demonstration of perfusion abnormalities discovered by MRI. We tried to find any distinctive feature of perfusion MRI, and described it in the manuscript. Also, we described some other conditions like non-vascular conditions as a differential diagnosis in such cases. We tried to improve the quality of written English in this manuscript. The following responses have been prepared to address all of the reviewers’ comments in a point-by-point fashion.

Reviewer 1

General Comments
This paper presents two cases of dural arteriovenous fistulas (dAVFs) presenting as neurological ischemic deficits. This form of presentation is not totally uncommon for dAVFs, as they can present with focal neurological deficits without visible hemorrhage in CT or MR. Therefore, the object of the paper is not completely unusual or rare. The most important aspect of the paper is the use of perfusion MR for the diagnosis of dAVFs. However, it is clear that this technique is not used for diagnosis of the dAVFs, as they were eventually discovered by the presence of a hemorrhage in one case, and by the presence of flow voids in MR in the second case. It is not clear whether these perfusion abnormalities were present in control studies after treatment of the dAVFs, as some authors describe the reversibility of the perfusion abnormalities after dAVFs treatment.

Authors’ reply. Authors would like to thank Reviewer “1” for the considering our manuscript interesting and reconsideration after revisions. We have incorporated the following specific comments in preparation of revised version of manuscript.

Specific Comments
1. The authors should improve their description of perfusion abnormalities in MR as well as describe whether these abnormalities were reversed by treatment or not.

Authors’ reply. The authors agreed on your comment about the detailed description of perfusion abnormalities. We analyzed the pattern of perfusion abnormalities in terms of MTT and rCBV (relative cerebral blood volume), which shows additional finding that the rCBV is increased in the area of MTT delay. This finding would strengthen the reported hypothesis that the venous
congestion is the main pathomechanism of transient symptoms.[1, 2] So, we added detailed description of perfusion abnormalities in the manuscript.

The authors also regret that the follow-up MRI was not taken to identify the reversibility of the perfusion changes after treatment.

Table. Parameters of perfusion weighted MRI.

<table>
<thead>
<tr>
<th></th>
<th>MTT (ms)</th>
<th>rCBV (a.u)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Case 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affected Side</td>
<td>1742.0±158.6</td>
<td>168.7±69.3</td>
</tr>
<tr>
<td>Contralateral Side</td>
<td>1621.7±24.6</td>
<td>161.6±92.1</td>
</tr>
<tr>
<td>Interhemispheric ratio</td>
<td>1.07</td>
<td>1.04</td>
</tr>
<tr>
<td><strong>Case 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affected Side</td>
<td>1512.9±48.7</td>
<td>195.2±94.4</td>
</tr>
<tr>
<td>Contralateral Side</td>
<td>1449.4±98.1</td>
<td>157.1±105.8</td>
</tr>
<tr>
<td>Interhemispheric ratio</td>
<td>1.04</td>
<td>1.24</td>
</tr>
</tbody>
</table>

MTT, mean transit time; rCBV, relative cerebral blood volume; a.u, arbitrary unit.

References

2. The authors should improve their paper as the main finding in their paper is the abnormality in perfusion discovered by MR and not the clinical presentation of their cases as this is not really unusual. Maybe they could determine if perfusion abnormalities correlate or not with the angio-architecture of the dAVF or not.

Authors’ reply. The authors agreed and tried to concentrate on the imaging findings in our reported cases. We also tried to describe the detailed angio-architecture of the dAVF correlating with the PWI abnormalities in the Discussion session.

Reviewer 2

General Comments
The authors report two cases with clinical presentation typical for transient ischemic attack (TIA), but with the dural arteriovenous fistula as an underlying mechanism. This paper illustrates wide spectrum of TIA mechanisms and TIA mimics, as well as a need for comprehensive and meticulous investigation, including multiparametric MRI, in many patients with TIA.

Authors’ reply. Authors would like to thank Reviewer “2” for the considering our manuscript interesting and your comments, which will improve the quality of this manuscript. Additionally, we acknowledge you for your meticulous suggestions of the English editing, which also improve the quality of this
manuscript. We have incorporated the following reviewer’s specific comments and suggestions of the English editing in preparation of revised version of manuscript.

Specific Comments

1. Regarding Case 1, could you please just briefly specify in the manuscript if neurological deficit (mild left hemiparesis and hemihypaesthesia) fully recovered two weeks after the dAVF embolization or some mild deficit remained, considering the size of ICH. Also, there is also no referral to the image B in the text.

Authors’ reply. Authors agreed that the paragraph can be misreading as if the patient was completely recovered, and text revised as per suggestions to avoid confusion. Also, we describe the imaging findings of figure B elsewhere.

2. Regarding Case 2, please state the duration of follow-during which patient remained symptom free.

Authors’ reply. The clinical follow-up in case 2 was for 6 months to our initial submission to BMC neurology, and text revised as per suggestions.

3. I believe the readership would benefit from short comment in the Discussion part of the manuscript on typical clinical and neuroradiological presentation of dAVF. This would significantly increase the informative value of the paper.

Authors’ reply. The authors agreed and text revised as per suggestions.

4. There are numerous publications in last few years on the subject (e.g. Ghobrial et al, World Neurosurg 2012, Morris, Radiol Clin North Am 2012, etc) so I kindly suggest the authors to consider adding at least one since 2009, which is the newest reference in the paper.

Authors’ reply. The authors agreed and text revised as per suggestions.

Reviewer 3

General Comments

This is a very interesting paper, which describes two patients with a TIA mimic due to a dural arteriovenous fistula. They conclude that isolated perfusion deficits often seen in TIA patients might also be due to a venous congestion.

Authors’ reply. Authors would like to thank Reviewer “3” for the considering our manuscript interesting and your comments, which will improve the quality of this manuscript. We have incorporated the following reviewer’s specific comments in preparation of revised version of manuscript.

Specific Comments

1. It has been reported that increasing the magnetic field strength allows the detection of additional small bright dots on DWI in TIA (Bertrand et al.
2008). Therefore, the authors should also give some information concerning MRI technique: Did they perform MR imaging on a 1.5 or 3.0 MR unit?

**Authors’ reply.** In our reported two cases, MR imaging was performed in 3.0T machine. Authors agreed on your comments about the description of MRI tesla and text revised as per suggestion in Case Presentation session.

2. Did the authors also perform a MR angiography in the first work-up after the first episode of case 1?

**Authors’ reply.** The authors also agree on your concern that the initial MR angiography might give some information about the dural AVF. To avoid any confusion to readers, we added paragraph describing the negative study of MRA in both cases.

3. Fig G: The signal void signs are probably temporal and not parietal.

**Authors’ reply.** The authors thought that the signal void signs are located between the central sulcus and the postcentral sulcus, so the location is parietal. We add serial sequences of GRE to help identify the location of the signal void signs.

Figure. The serial sequences of GRE in case 2.

4. The authors conclude that the transient deficits might be due to venous congestion. This implicate that venous congestion might also be transient. What might be the reason of a transient venous congestion?

**Authors’ reply.** To our knowledge, the relationship between the degree of venous congestion and the duration of neurologic deficits has not been proven in the literature. The authors only hypothesized that the degree of venous congestion can change depending on the degree of metabolic demand or changes in local intracranial and/or venous pressure, which can be associated with the transiency of neurologic deficits.