EDITORIAL

Constant facial pain in the trigeminal distribution. Does it respond to microvascular decompression?

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The article “Preoperative magnetic resonance imaging in Type 2 trigeminal neuralgia” by Burchiel and his colleagues1 in this issue of JNS is a logical continuation of the excellent work of this group on trigeminal neuralgia (TN). Their important contributions to this area include: a simple and practical classification of trigeminal pain into Type 1 (sharp paroxysmal pain) and Type 2 (constant dull and frequently burning pain); the refinement of MR imaging techniques to demonstrate the presence or absence of vascular compression at the root entry zone of the trigeminal nerve; the confirmation that the results of microvascular decompression (MVD) in patients with predominantly Type 2 TN are not satisfactory and that those who have only constant pain are very unlikely to get relief from the operation; and the fact that patients with pure Type 1 pain are much more likely to have vascular compression at surgery than those with predominantly Type 2 pain.

In this retrospective study these authors have attempted to answer the question of whether high-resolution MR imaging with 3D reconstruction can help select patients with Type 2 pain who are likely to respond to surgery and, in addition, to measure separately the response of the Type 1 pain compared with Type 2 pain to MVD. To this effect they reviewed retrospectively all patients who had constant pain at least 50% of the time (Type 2 TN by the authors’ classification) who had undergone high-resolution 3-T MR imaging with 3D reconstruction prior to surgery. Obviously, the ideal scientific study would have been to include all these patients and operate on all of them regardless of the MR imaging findings and then see if their outcome correlated with the preoperative MR imaging findings; in other words, did the patients who were predicted by MR imaging to have vascular compression indeed have vascular compression confirmed at surgery and did they do better than those who were predicted not to have vascular compression by the preoperative MR imaging? The problem with this study would have been that from previous experience Dr. Burchiel has gained the impression that when no vascular compression was predicted by MR imaging, it is unlikely that such compression would be found at surgery and it is unlikely that these patients would respond to the operation; therefore, it would have been unethical for him to subject to surgery the patients with Type 2 pain who were found to have no evidence of vascular compression on the preoperative MR images. Consequently, of the 27 patients in whom the aforementioned selection criteria (Type 2 pain and an adequate preoperative MR imaging) were met, 13 were selected for MVD on the basis of the clinical history and the prediction of vascular compression by the preoperative MR images. The other 14 patients were managed conservatively and were not studied further. Although not clearly stated, I presume that the features that the authors looked for on the clinical history to recommend surgery were the presence of sharp pain at some time as well as some of the other typical features of Type 1 pain such as memorable onset, response to anticonvulsants, trigger points, and others. In fact, Table 1 indicates that all of the 13 patients selected for surgery had Type 1 pain at some point and 69% of them still had it at the time of surgery.

The results are not surprising to this reviewer, and I am sure that they were not surprising to the authors either. At the time of surgery, definite neurovascular compression was confirmed in 11 of the 13 surgically treated patients. The facial pain was completely relieved in only 23.3% of these 13 patients, and it was not improved at all in an equal number; the rest (53.8%) experienced some degree of improvement. The most striking finding was that the sharp paroxysmal pains (Type 1) were relieved in all 10 patients who had such pain at the time of the operation. Conversely, the constant pain (Type 2) was improved (in some cases only minimally) in only 7 of the 13 patients, and only 3 of these patients had complete relief of their pain at the time of follow-up.

This is a very small study, it is retrospective, and, for the reasons discussed above, it is not controlled and therefore is subject to all the usual biases in such studies. Furthermore, because there is no objective way of measuring pain in these patients, both a placebo effect as well as a desire to please their surgeon could have been operative in the patients’ subjective reporting of their result. Nevertheless, in spite of these limitations, I believe that this study is important. The study confirms the value of the MR imaging technique that these authors have refined to predict the presence of vascular compression at surgery. More importantly, the authors have analyzed, I believe for the first time in a careful fashion, the differential response to MVD of the type of pain in patients who have both sharp and constant pain. Their data, based on small numbers, clearly suggests that the sharp pain is much more likely to respond to MVD than the constant pain.
Why is the sharp pain so much more likely to respond to MVD than the constant pain? In a very simplistic sense, I tend to think that the reason is that these 2 types of pain, although probably having a common origin (vascular compression), may have a different pathophysiology. It may be that sharp pains are due to early and reversible demyelination whereas the constant pain may be the result of more severe neuropathy perhaps involving irreversible axonal damage. This is likely to be the case in most patients in whom the syndrome begins with typical intermittent sharp lancinating pains and then may gradually develop, with time, some elements of constant dull burning pain. Perhaps the minority of patients in whom the syndrome starts with constant pain and in whom a sharp pain was always absent have a different etiology and may never have had vascular compression, as has been suggested by Dr. Burchiel’s previous work.

Finally, how will I, as a clinician, use the information from this study? Certainly this study does not tell us that the patients not selected for surgery on the basis of either not having any of the typical features of TN by history and/or not having evidence of vascular compression on preoperative MR imaging would not have done well had they only undergone surgery. However, I certainly would not operate on these patients because there is plenty of anecdotal evidence, including previous writings from this group, that these patients do not fare well with MVD. The take-home message from this study for me is that in patients with a good clinical history of typical TN but with some atypical features, such as a predominant component of constant pain, high-resolution MR imaging with 3D reconstruction can be helpful in selecting for surgery only those patients from this group who appear to have vascular compression in such a study. I will continue to offer MVD, regardless of MR imaging findings to all suitable surgical candidates with typical TN (Burchiel Type 1) and will not offer surgery to patients with Type 2 pain who do not have also sharp paroxysmal pain and some of the other typical features of TN. In this respect, I would use MR imaging to help with surgical selection in the same manner that, for example, I use CSF flow studies in patients with a Chiari malformation in whom I cannot be sure whether the symptoms, usually headaches, are due to the Chiari malformation or another source; if there is a significant block in the flow study I offer decompression to these patients, but otherwise I do not. Likewise, in patients with suspected normal-pressure hydrocephalus with a questionable history, I use ancillary studies, such as a spinal tap, to help me with surgical selection, but if the history is typical, I proceed with shunt surgery without ancillary studies.

We thank Dr. Burchiel and his colleagues for continuing to advance our understanding of TN and for offering us an important tool to select for surgery those patients with constant trigeminal pain who are more likely to respond to MVD.

Disclosure

The author reports no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Reference


Response

Kim Burchiel, M.D., Andrew Zacest, M.B.B.S., Stephen T. Magill, B.S., Jonathan Miller, M.D.

Dr. Heros’ reading of our paper, as stated in his commentary, exactly matches our thinking on how to apply the information in this article. He has also correctly pointed out that our study suffers from its retrospective nature, and the fact that we could not verify postoperative outcome in those patients in whom we made a decision to not offer MVD based on their MR imaging findings. That is, for patients with Type 2 pain, we did not offer surgery if MR imaging did not show definite neurovascular compression.

Our model of TN is really that it is a syndrome of 2 distinct pains, as Dr. Heros recognizes. This model would theorize that Type 1 pain is related to hyperactivity, possibly afterdischarges, in demyelinated larger axons, which “ignites” a wave of depolarization in the ganglion via cascading neurotransmitter release in the ganglion, as hypothesized by Devor and colleagues. These pains are triggerable by stimulation of the receptive fields of these injured axons and are felt as “electrical” or “lancinating.” Anticonvulsant agents can help alleviate these pains because they directly suppress hyperactive discharges from demyelinated or injured axons. It is probably this mechanism that explains why denervation of the trigger zone (radiofrequency lesion, radiosurgery, glycerol injection, balloon compression) is effective in relieving trigeminal neuralgia, whereas ablative procedures are rarely effective in other neuropathic pain states. The trigger for afterdischarges in the nerve is interrupted.

Type 2 pains are more like typical neuropathic pains elsewhere in the body, in that they are described as “burning,” “constant,” “dull,” or “aching.” These pains may be due, in part, to deafferentation from more severe and longstanding neurovascular compression. These pains likely have an origin that is central to the first-order trigeminal neurons. They are also much more difficult to treat, akin to neuropathic pains in general, and anticonvulsant agents are only marginally effective at best.

This model of trigeminal neuralgia helps us understand its mechanism and potential treatments for the pain. Subdivision of the pain types should allow better outcome and natural history studies. There are still many mysteries about this condition, which further study may penetrate. For example, why is it that in a substantial fraction of patients with otherwise typical (Type 1) TN, no neurovascular compression can be convincingly found by

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