Commentary for NEU-D-17-00877R1, titled "Trigeminal Nerve Compression without Trigeminal Neuralgia: Intraoperative vs. Imaging Evidence"

There is increasing interest in trigeminal neuralgia its pathophysiology, phenotype and imaging. Increasingly MRI's are being used as evidence that neurovascular contact (NVC) of the trigeminal nerve is present and that a microvascular decompression (MVD) is therefore a procedure that is indicated for relief of pain. Overall MRIs have high sensitivity but low specificity; however dislocation or atrophy of the nerve raises the specificity as does the exact location of the contact. Data is available from MRI studies which show that NVC is found in asymptomatic individuals but not dislocation or atrophy. Furthermore a recent consensus paper on a new classification and diagnostic grading system for TN proposes that if the MRI shows NVC with morphological changes of the trigeminal root that the condition should be named classical TN whereas those patients in whom the MRI shows no such changes should be named idiopathic TN [1]. Maarbjerg et al [2] go on to say that severe NVC is involved in the aetiology of TN and that this group of patients do have the clinical characteristics of TN. In this study surgical findings are noted in that patients with NVC of the facial nerve do exhibit neurovascular contact with the trigeminal nerve but no distortion or atrophy of the trigeminal nerve and no pain. These studies, therefore suggest that patients with these MRI characteristics should undergo MVD and will become pain free. Yet from reviews of the literature on MVD there is a consistent failure to get pain relief in 20-30% of patients. These results could be explained by several factors but one of these could be due lack of interdisciplinary consensus on both interpretation of the MRI scans and that of the operative findings. It could also be because the aetiology is not solely that of neurovascular compression. Studies using diffusion tensor imaging DTI and resting functional MRIs are suggesting that not only are there structural changes but connectivity varies and these are altered by radiofrequency rhizotomy. [3]

We still have no biomarkers and need to rely on the patients' subjective history. Studies are increasingly showing that the phenotype is not as simple and attacks are not as stereotypic as previously postulated [4, 5, 2]. There is general consensus that some patients with TN also have a more continuous background pain and this group also have neurovascular compression. Studies show that increased frequency and duration of paroxysms are not linked with duration of disease nor to MRI findings. There is thus a need to phenotype patients carefully, perform high quality MRI, DTI and resting functional MRIs and correlate these with surgical findings and subsequently outcomes. This needs multidisciplinary teams working across several centres which do a range of surgical procedures.

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