Microvascular Decompression for Trigeminal Neuralgia: The Role of Mechanical Allodynia

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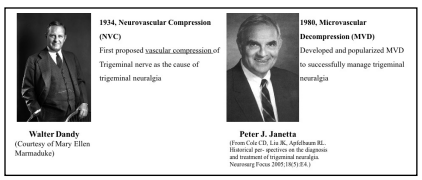
Introduction

MVD is ranked as the most effective remedy for TN. Nevertheless, not all patients can be completely cured by MVD, and recurrence or delayed relief may occur in a small proportion of patients. This dilemma reflects the lack of thorough understanding TN mechanisms, which are now considered a unique form of neuropathic pain and the most common type of neuralgia. This study was conducted to determine whether mechanical allodynia (MA) acts as a predictor of outcome after microvascular decompression (MVD) for trigeminal neuralgia (TN) and discuss the potential pathological mechanisms involved.



Methods

A series of 246 patients who underwent MVD for TN were involved in the study. The classifications were based on the characteristic of pain (shock-like or constant), and the presence of MA was defined from the chart review, retrospectively. The surgical outcome is defined as excellent, good, and poor. Immediate and long-term outcomes were compared to provide the information on recurrence and delayed relief. The relationship among the groups was investigated, and the strength was determined.

Table 2. The Results of Immediate Outcome After Microvascular Decompression

Classification	Excellent	Good	Poor
MA (n, %)	115 (61)	42 (22)	31 (17)
NMA (n, %)	15 (26)	18 (31)	25 (43)
T1TN (n, %)	120 (58)	48 (23)	40 (19)
T2TN (n, %)	10 (26)	12 (32)	16 (42)

MA, mechanical allodynia; NMA, non-MA, absence of mechanical allodynia; DM, diabetes mellitus; T1TN, type 1 trigeminal neuralgia; T2TN, type 2 trigeminal neuralgia.

Table 3. The Results of Long-Term Outcome After Microvascular Decompression

Classification*	Excellent	Good	Poor	Recurrence	Delayed Relief
MA (n, %)	116 (61)	35 (19)	37 (20)	18 (10)	14 (7)
NMA (n, %)	12 (21)	13 (22)	33 (57)	15 (26)	6 (10)
T1TN (n, %)	118 (57)	38 (18)	52 (25)	26 (13)	15 (7)
T2TN (n, %)	10 (26)	10 (26)	18 (48)	7 (18)	5 (13)

MA, mechanical allodynia; NMA, non-MA, absence of mechanical allodynia; DM, diabetes mellitus; T1TN, type 1 trigeminal neuralgia; T2TN, type 2 trigeminal neuralgia.
*The classification is based on the initial review of medical history chart.

Results

Both presence of MA and type of TN pain are significant predictors of surgical outcome ($P < 0.05$). MA was proven to be an independent predictor of surgical outcome and also a significant predictor of existence of neurovascular compression ($P < 0.05$) and lower rate of recurrence ($P < 0.05$). No statistically significant predictors of delayed relief were detected in this study.

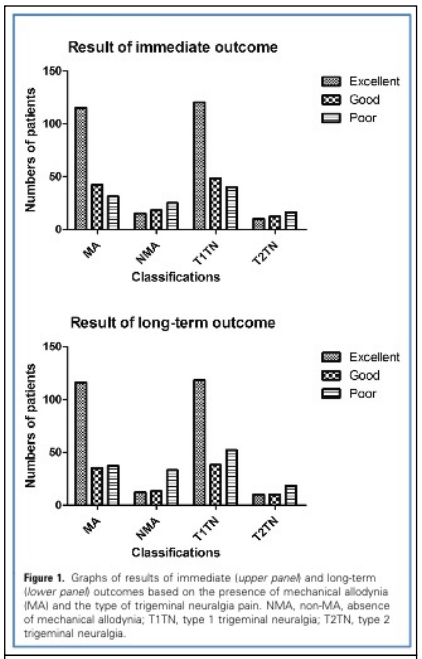


Figure 1. Graphs of results of immediate (*upper panel*) and long-term (*lower panel*) outcomes based on the presence of mechanical allodynia (MA) and the type of trigeminal neuralgia pain. NMA, non-MA, absence of mechanical allodynia; T1TN, type 1 trigeminal neuralgia; T2TN, type 2 trigeminal neuralgia.

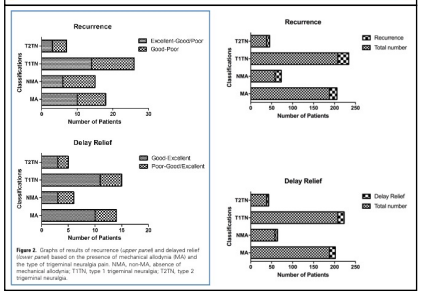


Figure 2. Graphs of results of recurrence (*upper panel*) and delayed relief (*lower panel*) based on the presence of mechanical allodynia (MA) and the type of trigeminal neuralgia pain. NMA, non-MA, absence of mechanical allodynia; T1TN, type 1 trigeminal neuralgia; T2TN, type 2 trigeminal neuralgia.

Conclusions

The presence of MA is a reliable predictor of immediate and long-term outcome after MVD for TN. Compared to the patients without MA, the incidence rate of intraoperative neurovascular compression (NVC) was higher in MA-positive patients, who were more likely to achieve a better outcome and lower rate of recurrence after MVD for TN. Application of the information in this study will be helpful in patient selection of MVD for TN.

Learning Objectives

By the conclusion of this session, participants should be able to:1)describe the importance of mechanical allodynia in predicting the surgical outcome of trigeminal neuralgia;2)discuss the relative mechanism involved in trigeminal neuralgia based on the recent progress in basic research on pain.

References

[1]Dandy WE. Concerning the cause of trigeminal neuralgia. Am J Surg 1934;24:447-455.
[2]Jannetta PJ, Robbins LJ. Trigeminal neuropathy--new observations. Neurosurgery 1980;7(4):347-51.
[3]Cruccu G, Gronseth G, Alksne J, Argoff C, Brainin M, Burchiel K, Nurmikko T, Zakrzewska JM, American Academy of Neurology S, European Federation of Neurological S. AAN-EFNS guidelines on trigeminal neuralgia management. European journal of neurology : the official journal of the European Federation of Neurological Societies 2008;15(10):1013-28.
[4]Burchiel KJ. A new classification for facial pain. Neurosurgery 2003;53(5):1164-6; discussion 6-7.
[5]Sandkuhler J. Models and mechanisms of hyperalgesia and allodynia. Physiological reviews 2009;89(2):707-58.
[6]Campbell JN, Raja SN, Meyer RA, Mackinnon SE. Myelinated afferents signal the hyperalgesia associated with nerve injury. Pain 1988;32(1):89-94.
[7]Sandkuhler J. Understanding LTP in pain pathways. Molecular pain 2007;3:9.
[8]Todd AJ. Neuronal circuitry for pain processing in the dorsal horn. Nature reviews. Neuroscience 2010;11(12):823-36.
[9]McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. Journal of neurosurgery 1999;90(1):1-8.
[10]Battista AF, Alban E. Effect of graded ligature compression on nerve conduction. Experimental neurology 1983;80(1):186-94.
[11]Basbaum AI, Bautista DM, Scherrer G, Julius D. Cellular and molecular mechanisms of pain. Cell 2009;139(2):267-84.