The Incidence of Vascular Conflict with the Trigeminal Nerve in Cluster Headache

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INTRODUCTION

Although neurovascular conflict is implicated in facial pain syndromes such as trigeminal neuralgia, its role in Cluster Headache (CH) has not been systematically reviewed. In this study, we assess the incidence of radiographic arterial and venous conflict with the trigeminal nerve in symptomatic versus asymptomatic trigeminal nerves in a cohort of patients with CH.

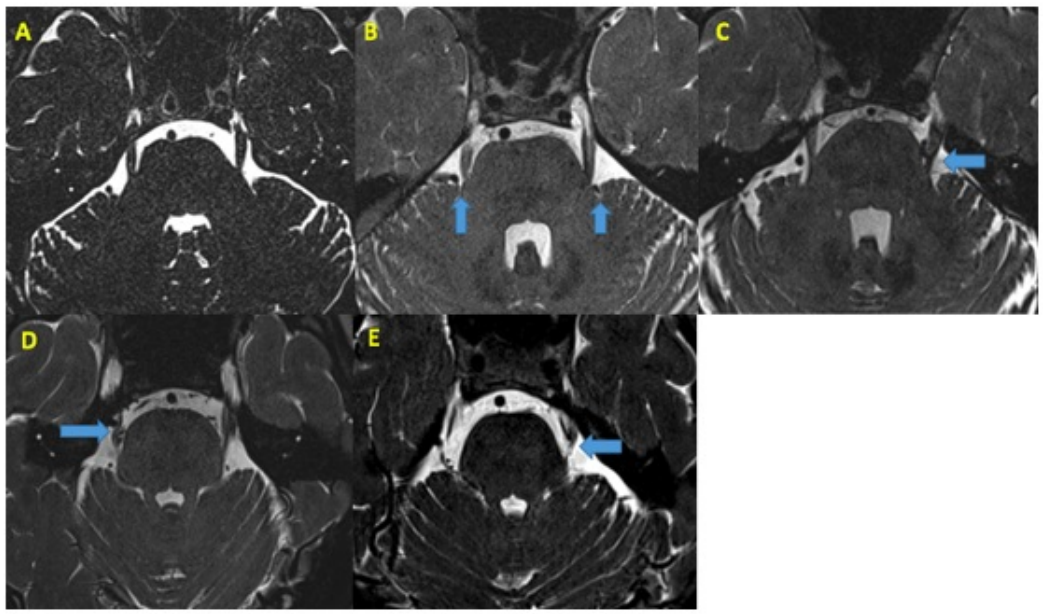

Figure 1: High resolution magnetic resonance (MRI) images demonstrating the relationship of adjacent vasculature to the trigeminal nerves in CH patients. **A**: Group (1) no nearby vessel; **B**: Group (2) vessel proximity; **C**: Group (3) vascular contact; **D**: Group (4) distortion; **E**: Group (5) atrophy.

METHODS

Patients with confirmed CH at a single institution underwent high-resolution MRIs of the prepontine cistern between 2007 and 2017. Prospectively acquired images were retrospectively reviewed by a neuroradiologist and two neurosurgeons blinded to symptom severity and lateralization. Each trigeminal nerve was classified into one of the following groups based on its relationship to adjacent vasculature: (1) no vessel within 1 mm, (2) vessel within 1 mm, (3) vascular contact, (4) vascular distortion of the nerve and (5) vascular contact with nerve atrophy or signal change.

RESULTS

136 patients (mean age at imaging: 44.5 years, mean age of symptom onset: 31.0 years) had 153 symptomatic and 119 asymptomatic nerves (55 left-sided; 64 right-sided; 17 side-alternating attacks). Incidence of arterial conflict (groups 3, 4 and 5) was significantly higher in symptomatic compared to asymptomatic nerves (40.5% vs. 26.9%, p=0.02). Incidence of venous conflict was not significantly different between symptomatic versus asymptomatic nerves (42.5% vs. 42.0%, p=0.94).

Incidence of Arterial Relationship with the Trigeminal Nerve in all Patients

	Symptomatic nerve N (%)	Asymptomatic nerve N (%)	P value
Total number	153	119	
1. No nearby artery	88 (57.5)	84 (70.6)	0.03
2. Arterial proximity	3 (2.0)	3 (2.5)	1.00
3. Arterial contact	40 (26.1)	18 (15.1)	0.03
4. Distortion	14 (9.2)	6 (5.0)	0.20
5. Atrophy	8 (5.2)	8 (6.7)	0.60
Arterial Conflict (3, 4 & 5)	62 (40.5)	32 (26.9)	0.02*

CONCLUSIONS

Arterial contact is neither required nor sufficient to drive CH attacks, but it may interact with a central (hypothalamic) generator to drive symptoms in a subgroup of patients. This suggests a rationale for microvascular decompression in refractory CH patients who have radiological arterial contact with the nerve on the symptomatic side.