



A Cadaver Study for Investigating Vascular Contact with the Trigeminal Nerve

Trigeminal Sinire Vasküler Temasın Araştırıldığı Bir Kadavra Çalışması

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ABSTRACT

Objective: The present study investigates root variations within the cisternal segment of the trigeminal nerve and the distribution of vascular compression at the root entry zone in a normal population.

Material and Methods: In total, 60 trigeminal nerves taken from 30 fresh cadavers, selected based on a simple random sampling method, were dissected with an arachnoid dissection from the exit point of the trigeminal nerve from the brainstem until the point of entry to Meckel's cave using a binocular loop and microsurgery set. The vicinity of the vascular structures and the types of trigeminal nerve in the cisternal segment were investigated.

Results: In total, 30 cases of vascular contact were detected, involving arterial and venous structures in 22 (73.3%) and 8 (26.6%) cases, respectively. Arterial structures were defined as the superior cerebellar artery and the anterior inferior cerebellar artery in 14 (46.6%) and 8 (26.6%) cases, respectively. The superior petrosal vein was found to be responsible for venous contact to the trigeminal nerve.

Conclusion: The results of this cadaveric study showed that vascular contact could be incidentally found in 50 percent of the normal population.

Key Words: Trigeminal nerve, Cadaver, Anatomical study, Vascular structures

ÖZ

Amaç: Trigeminal sinirin sisternal segmenti içinde kök varyasyonları, roor enter zone'a vasküler basının normal popülasyondaki dağılımını araştırmak.

Gereç ve Yöntemler: Basit rastgele örneklem metoduyla seçilmiş 30 taze kadavrada, 60 trigeminal sinir, trigeminal sinir beyinsapı çıkış noktasından, Mekcel's cave giriş noktasına kadar binoküler lup ile mikrocerrahi set kullanılarak araknoid diseksiyonu yapılmıştır. Sisternal segment içinde trigeminal sinirin ve vasküler yapılarla olan komşuluk ilişkileri ve tipleri araştırıldı.

Bulgular: 30 vasküler temasın saptandığı olgularda 22(%73,3)'sinde arter, 8(%26,6)'inde venöz yapılar görüldü. Arteriel yapıların 14 (%46,6)'ünde süperior cerebellar arter, 8 (%26,6)'inde anterior inferior cerebellar arter olarak tanımlandı. Süperior petrozal ven, trigeminal sinire venöz temastan sorumlu bulunmuştur.

Sonuç: Bu kadavra çalışmasının sonuçlarına göre vasküler temas insidental olarak normal popülasyonun %50'sinde görülmektedir.

Anahtar Sözcükler: Trigeminal sinir, Kadavra, Anatomik çalışma, Vasküler yapılar

INTRODUCTION

The trigeminal nerve (the fifth cranial nerve), being the largest and the thickest of all cranial nerves, develops embryologically from the first branchial arc. The trigeminal nerve, which arrives at a large ganglion known as the *ganglion trigeminale*, separates into three branches, known as the *nervus (n.) ophthalmicus* (V1), *n. maxillaris* (V2) and *n. mandibularis* (V3). After exiting the brainstem, the trigeminal nerve proceeds through the cisternal segment, Meckel's cave segment, trigeminal ganglion and peripheral segment (1). Its root and the *ganglion trigeminale* are in close proximity to important structures in the cranial fossa (2-5). The trigeminal nerve receives stimuli from a large part of the scalp, teeth, and oral and nasal cavities. It supplies sensation to the face and separates to the motor branches in the chewing muscles.

Trigeminal neuralgia (TN) is a facial pain syndrome that develops due to paroxysmal attacks – usually unilateral electrical discharges within the innervation region of the trigeminal nerve branches. Trigeminal neuralgia generally affects the elderly, and its incidence has been estimated at 4–5/400,000, with only 1 percent of TN patients being younger than 20 years (7-9). The most common cause of TN is to compress the nerve in the root enter zone (REZ) region of the vascular structures. This etiology was first described by Jannetta in 1967 (10), who went on to introduce microvascular decompression surgery for the treatment of TN, which develops due to vascular compression (11).

In the present study, we determine the distribution of vascular compression on REZ in a normal population by investigating 60 trigeminal nerves of 30 fresh cadavers through a transtentorial opening.

MATERIALS and METHODS

This article was performed by permission of the Presidency of Scientific Board of Council of Forensic Medicine (Approval number: B.03.1.ATK.0.01.00.08/ 622). A total of 30 fresh cadavers, including 21 males and 9 females aged 18 years or older, with no intracranial injuries, were obtained from the Forensic Medicine Institute Izmir Group Directorate Morgue Specialization Unit via a simple random sampling method. Cadavers associated with death due to cerebral pathologies were excluded from the study. It is not known whether the cadavers had trigeminal neuralgia in their history.

Autopsy Procedures

Ethical principles and dissection rules were considered during this study, and all autopsies were performed in the Autopsy Room of the Forensic Medicine Institute Izmir Group Directorate Morgue Specialization Unit. Skin lines were determined for skin dissection, and an interauricular

line was drawn on the frontal direction, passing from the vertex. This line was incised using a number 4 scalpel and the skin and subcutaneous tissues were incised. The calvaria was revealed by folding the skin and subcutaneous tissues behind the incision backwards and folding the part in front of the incision to the front. A vibrant dissection saw (Electronic Power Gipsage, Germany) was used to lift the calvaria (Figure 1A). After the calvaria was lifted, the cerebrum was pulled upwards by holding from the frontal region. The cerebrum was removed with an incision to the mesencephalon (tentorium level), allowing the visualization of the *n. trigeminus*. After the tentorium was cut, the arachnoid around the trigeminal nerve was dissected using a binocular loop (Ergonoptix 3.5 mm*420 mm) and a microsurgery set (Figure 1B). The course of the trigeminal nerve was investigated and neighboring structures (Figure 2B), variations and the relationship between the nerve and the vascular structures was noted and photographed using a Canon digital IXUS 70 7.1-megapixel camera (Figure 1C).

Classification of the Types of Relationship With Vascular Structures

In order to examine the contact between the trigeminal nerve and the vascular structures, the relationship between the sensorial and motor parts of the nerve and the position of the venous structures with respect to the trigeminal nerve were observed. Additionally, the types of contact between the vascular structures and the trigeminal nerve were recorded for each vascular structure as touching, compression and distortion, or embedding (Figure 2A-C).

RESULTS

Demographical Data of the Cadavers

The autopsy sample comprised 21 males and nine female cadavers. The mean age of the cadavers was 48.3 years (SD: 21.7), and the mean height and body weight were 170.0 cm and 68.9 kg, respectively. Causes of death were gunshot injury (6), sharp object injury (5), myocardial infarction (5), traffic accident (3), hanging (4), drowning (3), alcohol intoxication (1), lower gastrointestinal system bleeding (1) and cardiac tamponade (1). The duration between time of death and autopsy ranged between 12 and 24 hours.

Results of Anatomical Investigations

When the 60 trigeminal nerves from the 30 fresh cadavers were investigated, vascular contact was detected in the REZ region of 30 trigeminal nerves. The structures in contact with the trigeminal nerve were the superior cerebellar artery (SCA), the anterior inferior cerebellar artery (AICA) and the superior petrosal vein (SPV). The distribution of vascular contact was not different between the right and left trigeminal nerves, with 15 right and 15 left vascular

contacts in total. Anatomical investigation revealed no aneurysms, tumors or persistent trigeminal artery vascular malformations. The trigeminal nerve was not in contact with the basilar artery or aberrant pontine artery.

In total, 30 cases of vascular contact were detected involving arterial and venous structures in 22 (73.3%) and eight (26.6%) cases, respectively. The involved arterial structures were the SCA and AICA in 14 (46.6%) and eight (26.6%) cases, respectively. The SPV was responsible for venous contact, and three types of contact were observed between the REZ region of the trigeminal nerve and the vascular structures:

- Compression and distortion (5)
- Touching (19)
- Embedding (6), (Figure 2A)

Of all cases of vascular contact, 13 involved the sensorial root, eight involved the motor root and nine involved both the sensorial and motor roots (Figure 2C).

The relationship between the SPV and trigeminal REZ

was defined in five different forms as anterior (1), posterior (3), rostral (2), caudal (1) and rostral+caudal (1).

All the results of the study are presented in the Table I.

DISCUSSION

The precise etiology and pathophysiology of TN has not been clearly elucidated. There are many theories about the etiopathogenesis of TN. The most acceptable of these theories is the theory of ignition. According to this theory, a painful hyperexcitability status occurs in the axons as a result of abnormalities in the afferent neurons of the trigeminal nerve stem or ganglion. It has also been suggested that central sensitization plays a role in trigeminal neuralgia. In most of the cases, the reason is thought to be the demyelination of the root entry zone in the trigeminal nerve root. In many patients, this demyelination is the result of compression of the trigeminal nerve by vascular structures. Indeed, autopsy studies carried out on patients diagnosed with TN have shown demyelination and dysmyelination in nerve biopsies (12). The etiology of TN is known to involve neurovascular compression in 80–90

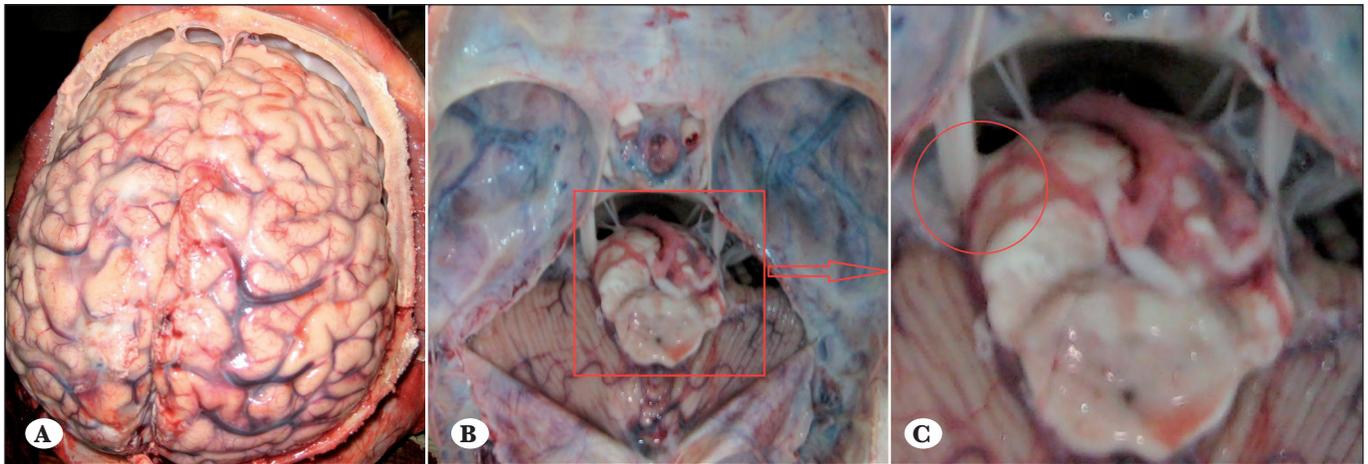


Figure 1: **A)** The cerebrum image after calvarium was removed. **B)** The cerebrum removed was removed from the mesencephalon level; bilateral trigeminal nerves and cysternal segment were observed. **C)** Touch type contact of the superior cerebellar artery with the superior surface of the left trigeminal nerve.

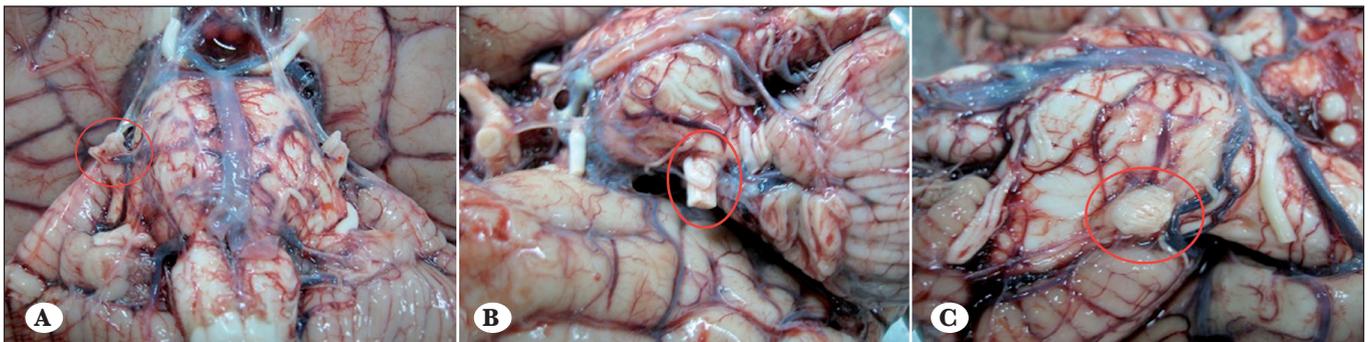


Figure 2: **A)** Trigeminal nerve, superior petrosal vein embedding type contact **B)** The trochlear nerve is seen in the upper part of the trigeminal nerve and there is no vascular contact. **C)** The right trigeminal nerve, motor and sensory fibers are seen in close proximity.

percent of the cases. In rare cases, a vascular compression, a saccular aneurysm or an arteriovenous malformation may be responsible for the etiology of trigeminal neuralgia, although TN etiology may also involve schwannomas, meningiomas, epidermoid cysts, or plaques of multiple sclerosis. Benign tumors originating from the bones, and deformities due to osteogenesis imperfecta have also been reported in the literature as very rare causes of TN (11,12).

In 1934, Dandy explained the cause of trigeminal neuralgia with a theory similar to vascular compression. Based on intraoperative findings, he reported that the trigeminal nerve was exposed to vascular compression in 45 percent of cases, and that the artery causing this compression was the AICA (13-15).

In 1977, Jannetta suggested that the trigeminal nerve root was compressed and distorted by an arterial fold or

Table I: Demographic and anatomical data of the study.

No	Death Cause	Sex	Age	Height cm	Weight kg	Arterial-Venous Touch (Left/right)	SPV Touch position	Vascular touch to sensor motor root (left/right)	Trigeminal n. and vascular relation type (left/right)
1	M.i	M	55	160	65	-/-	-	-	-
2	T.a	M	83	172	70	SCA/SCA	-	S+M/S	T/T
3	T.a	M	21	175	63	SPV/-	anterior	S+M	E
4	M.i	M	48	168	75	-/SCA	-	S	C.D
5	Ct.i	M	41	178	80	AICA/-	-	S	T
6	M.i	M	75	169	73	-/SCA	-	M	T
7	M.i	F	68	157	64	AICA/-	-	S	C.D
8	Fa.i	M	33	175	70	SCA/SCA	-	S+M/M	T/T
9	H.	M	66	166	60	SCA/AICA	-	S+M/S	T/T
10	D.	M	79	164	69	SPV/-	posterior	S	E.
11	T.a	M	50	170	65	AICA/-	-	S	T
12	H.t	F	60	158	62	-/SPV	caudal	S	T
13	D.	F	45	167	70	-	-	-	-
14	Ct.i	M	60	179	80	-	-	-	-
15	M.i	M	55	168	66	SPV/-	posterior	S+M	E.
16	Gs.b	M	50	167	60	AICA/-	-	S	T
17	A.i	M	26	180	85	AICA/SPV	rostral	S/S+M	T/E.
18	Fa.i	M	61	182	75	SCA/-	-	M	T
19	Fa.i	M	55	184	73	SPV/-	rostral+caudal	S+M	E
20	Fa.i	F	55	165	60	AICA/-	-	S	T
21	H.	F	38	170	68	-	-	-	-
22	Fa.i	F	45	161	59	-/SCA	-	M	T
23	Fa.i	F	37	164	71	-/SCA	-	M	T
24	D.	M	21	176	82	SCA/SPV	posterior	S+M/M	C.D/E
25	H.	M	52	166	51	SCA/SPV	rostral	S+M/M	C.D/T
26	Ct.i	F	37	165	52	-	-	-	-
27	H.	M	21	177	62	-	-	-	-
28	Ct.i	M	27	169	65	-/SCA	-	M	T
29	Ct.i	M	55	176	88	-/AICA	-	S	C.D
30	D.i	F	21	172	84	-/SCA	-	S	T

M.i: Myocard infarct, **T.a:** Traffic accident, **Ct.i:** Cutting tool injury, **Fa.i:** Firearm injury, **H.:** Hanging, **D.:** Drowning in water, **H.t:** Heart tamponade, **Gs.b:** Gastrointestinal system bleeding, **A.i:** Alcohol intoxication, **D.i:** Drug intoxication, **SCA:** Superior cerebellar artery **AICA:** Anterior inferior cerebellar artery, **SPV:** Superior petrosal vein, **Sens.,(S):** Sensory, **M:** Motor **T:** Touch **C.D:** Compression and distortion **E:** Embedding

a vein at the point of exit from the pons in TN patients who underwent subtemporal and suboccipital approach. Jannetta considered this compression and distortion to be an etiological factor, and proposed a “microvascular decompression” technique to remove the compression from the trigeminal nerve (16). Jannetta also highlighted that the compression should be at the “root entry zone” (REZ) in order to induce pain (16). In 1,185 patients who underwent microvascular decompression, Jannetta reported that the SCA and AICA was involved in 75 percent and 10 percent of the cases, respectively, while 68 percent of the cases had concurrent venous and arterial compression, and 18 percent had venous compression alone (17).

Haines et al. (18) compared and classified contact of the trigeminal nerve with vascular structures based on data obtained from 40 operated patients with TN and 40 trigeminal nerves from 20 cadavers, and identified vascular contact in 18 (45%) of the 40 REZs, 14 of which involved SCA and four with venous compression. The neurovascular contact type was in the form of touching and compression in 77 percent and 22.2 percent of the cases, respectively (18). The compression and distortion types of neurovascular contact, particularly on the axilla of the trigeminal nerve, have been suggested to have an impact on neuralgia (18). Evidence of venous compression was found in 10 percent of the cadavers and 20 percent of intraoperative observations. Contrary to Dandy, Hains et al. suggested that venous compression may play a role in the etiology of TN (18). In another study, Hardy and Rhoton detected vascular contact in 30 (60%) of 50 REZs from 25 cadavers, and of those contacts, 26 and four involved the SCA and AICA, respectively (19). In their cadaver study, Ramesh and Premkumar detected vascular contact in 39 of 100 REZs, and found that the SCA was involved in 23 (59%), the AICA was involved in 7 (17.9%), pontine branch of basilar artery was involved in four (10.2%) and venous contact was present in five (12.9%) cases. The reported contact types in this study were touching and compression-

distortion in 28 (71.8%) and 11 (28.2%) cases, respectively (20).

Since the 2000s, improvements in magnetic resonance imaging techniques and increases in specific sequences have allowed a more detailed visualization of the relationships between the trigeminal nerve and vascular structures, using in particular sequences such as 2D time of flight (TOF), 3D (TOF) and CISS (21). Accordingly, recent studies investigating the trigeminal nerve have been mostly radiological in nature. In an MRI study by Yoursry et al. (22), vascular contact was identified in 36 of 80 trigeminal REZs, and of those cases, 30 and six involved the SCA and AICA, respectively. Another radiological study demonstrated vascular contact in 80 of 286 trigeminal REZs, and reported the involvement of the SCA in 72 and the AICA in eight of those contacts (23).

There have been only a limited number of cadaver studies in the literature investigating the trigeminal nerve and its vascular contacts. The contact of the trigeminal nerve REZ and arterial structures is more common. As seen in all anatomical and radiological studies, among all arterial structures, the SCA-REZ relationship is responsible for more than half of all compressions. Consistent with the findings of previous studies, the results of the present study supported the widely accepted theory of vascular compression in the etiology of trigeminal neuralgia.

Based on our literature review, this is the first autopsy study to be carried out in Turkey reporting on the relationship between the trigeminal nerve and vascular structures.

Although the trigeminal nerve is in contact with the vascular structures in almost 50 percent of cases, the fact that TN is predominantly a disease of the elderly population with an incidence of 1/100,000 underlines the importance of future studies to clarify contact types and any secondary causes that increase with age, such as atherosclerosis leading to vascular elongation.

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