

Frequency and Etiologies of Peripheral Type Cranial Neuropathy in the Neurology Inpatient Clinic

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ABSTRACT

Introduction: Peripheral type cranial neuropathies (PCNP) other than idiopathic peripheral facial paralysis are quite rare. In this study, we analysed patients with PCNP who were treated in our inpatient clinic.

Methods: The patients who were hospitalized, examined and treated in our clinic between 1/1/2018 and 31/12/2023 were examined retrospectively. Sixty-three (34 men) out of total 3593 patients were found to have isolated PCNP. Demographic data, aetiologies, cerebrospinal fluid (CSF) findings, treatments and course of these patients were documented.

Results: The average age of the patients was 56.25±15.55 years. Forty-three patients had single (unilateral/bilateral) and 20 had multiple PCNP. The most common involvement was seen in the VI. cranial nerve (24 isolated, 16 with other cranial neuropathies). The most common etiology was autoimmune/inflammatory (25 patients), followed by peripheral nerve ischemia (23 patients), infectious (6 patients), and tumoral infiltration

(5 patients). Two patients with multiple PCNP had tumoral infiltration, Herpes infection in 2, COVID-19 in 2, Brucellosis in 1, and tuberculous meningitis in 1. Ischemic causes were most commonly associated with diabetes-related microvascular damage. Magnetic resonance imaging (MRI) examination revealed tumoral infiltration in 5 patients. Other MRI findings were cavernous sinus involvement and contrast enhancement of cranial nerves. CSF was examined in 53 patients; CSF protein was high in 20; pleocytosis was observed in one.

Conclusion: Among peripheral cranial nerves, VIth is the most commonly involved, alone/together with other cranial nerves (CN). Inflammatory causes most commonly play a role in the etiology of PCNP. Malignancy and infectious etiologies should be considered first in patients with multiple cranial neuropathies (CNPs).

Keywords: Abducens paralysis, cranial neuropathy, facial paralysis, inflammatory neuropathy

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INTRODUCTION

There are 12 pairs of CNs, six on the right and six on the left. Three of the CNs (I, II and VIII) are involved only in specialised senses such as smell, vision, hearing and balance, respectively. Five (III, IV, VI, XI and XII) are pure motor nerves. The other four (V, VII, IX and X) have motor and sensory functions, which are classified as mixed nerves (1). Four CNs (III, VII, IX and X) also contain parasympathetic fibres that are associated with autonomic functions (1). The first two CNs originate in the cerebrum and the remaining 10 in the brain stem which extend beyond the central nervous system (CNS) through foramina in the skull base.

Cranial neuropathies (CNPs) can result from any pathologies affecting nerve fibres at any point from the nuclei of the CNs to the end organ to which the nerve supplies (2,3). Lesions distal to the nucleus of the CNs (peripheral CN; PCNP) can cause isolated CN involvement, whereas pathological processes affecting the brain, the tracts to the CN or the CN

nuclei are often associated with multiple neurological symptoms. Some of the CNs follow long, circuitous routes to reach their end organs. A detailed knowledge of CN anatomy combined with a careful neurological examination is required for accurate clinical localisation of potential lesions and selection of appropriate imaging protocols.

PCNPs in adults may develop due to many causes including inflammation, infection, trauma, microvascular ischaemia and other vascular events, tumoral infiltration, externally compressing masses and toxic causes (4). The most common PCNP is the VIIth PCNP due to Bell's palsy with an annual incidence of 20 per 100.000. In addition, diabetes mellitus (DM) and hypertension (HT), which are commonly observed in the general population, may cause PCNP through microvascular ischaemia; and CNs III, VI and IV are frequently affected, in that order (5). Multiple PCNPs are clinical pictures observed with concomitant

Highlights

- Vth CN was the most frequently affected in PCNP cases.
- Inflammatory PCNP cases increased significantly after the Covid-19 pandemic.
- Tolosa-Hunt syndrome and GBS were key inflammatory causes of PCNP.

or sequential damage of two or more CNs. Infections involving the meninges, tumoral infiltrations and inflammatory diseases such as sarcoidosis play a role in the etiology (6).

During the Covid-19 pandemic, CNP isolated or associated with Guillain-Barre syndrome (GBS) was frequently reported. In the review of Finsterer et al., published in 2022, it was found that the CNs VII, VI and III were most frequently involved, respectively (7). In this study, we aimed to make a detailed analysis of patients with PCNPs due to lesions outside the brain parenchyma, who were examined and treated as inpatients in a tertiary neurology clinic, to examine the etiological causes and to investigate the effect of Covid-19.

METHODS

Ethics Committee Approval and Evaluation of the Patients

This retrospective cross-sectional study was approved by the Bezmialem Vakif University Clinical Research Ethics Committee (No: E-54022451-050.04-149093, Meeting date: 26.04.2024). Between January 1, 2018 and December 31, 2023, files of 3593 patients who were hospitalised, examined and treated in the neurology inpatient clinic were retrospectively examined. We detected 63 patients (34 males) with PCNP without any lesion in the brain parenchyma on magnetic resonance imaging (MRI). Olfactory dysfunctions and inflammatory optic neuritis were excluded.

Demographic data (age, gender), complaints and history, other comorbidities, neurological examination findings, investigations for the etiology [imaging, cerebrospinal fluid (CSF) and other laboratory findings], treatments and disease outcomes of the patients were documented. The characteristics of the cases before and after March 11, 2020, the date of diagnosis of the first Covid-19 case in our country, were compared.

Peripheral type cranial nerve (PCN) involvements are grouped as single or multiple. Etiologies were classified as inflammatory, ischaemic, tumour infiltration, infectious and trauma. The ischemic etiology was determined based on the presence of significant vascular risk factors (such as HT, DM, and other cardiovascular risk factors) and MRI findings consistent with small vessel disease (e.g., white matter hyperintensities, periventricular or deep subcortical ischemic changes). Additionally, CSF analysis did not reveal any findings suggestive of alternative etiologies, such as inflammatory, infectious, or neoplastic causes. Based on these clinical, radiological, and laboratory findings –and in accordance with previously established criteria for microvascular cranial neuropathies– an ischemic origin was considered the most likely explanation for these cases.

The presence of comorbid malignancy, stroke, HT and DM were recorded. Findings of detailed neurological examination including cranial nerve functions, motor, sensory and balance evaluation were recorded

as normal or abnormal. Brain MRI findings were recorded as normal or abnormal. Among CSF findings, cell count was recorded as cells/mm³ protein, angiotensin converting enzyme (ACE) levels were recorded as normal or elevated, and oligoclonal bands (OCB) were recorded as present or absent. Vasculitis markers were evaluated as normal or abnormal.

Treatments are classified as intravenous immunoglobulin (IVIg), corticosteroids, plasmapheresis, antibiotherapy and other. Therapeutic outcome was graded as spontaneous recovery, no response, partial or complete response.

Statistics

Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) Software program (version 25.0; IBM, Armonk, NY, USA). Descriptive statistics for continuous variables were presented as mean ± standard deviation (SD) and categorical variables were given as ratio (%).

RESULTS

PCNP was detected in 1.75% (63 patients) of the total 3593 patients hospitalised in our neurology clinic in a period of 72 months. Thirty-four of these patients were male and 29 were female with a mean age of 56.25±15.55 (range: 18–78) years. Forty-three patients had involvement of a single PCN (unilateral/bilateral) and 20 patients had more than one different PCNP (Table 1). The Vth CN was the most commonly involved single nerve (24 of 43 patients; 55%), followed by the VIIth and IIIrd CNs [6 patients (14%), 6 patients (14%), respectively].

In the medical history of the patients, 27 had DM, 30 had HT, 4 had malignancy, 6 had stroke, and 10 had other comorbidities. Fifteen patients had no known history of comorbidities.

Other findings accompanying PCNP on neurological examination were as follows: 1 patient had a motor deficit, 1 patient had a superficial sensory deficit other than the involved PCN, and 22 patients had deep sensory deficit. The weakness and sensory deficit of 1 patient with motor and superficial examination findings were sequelae and developed after lumbar fracture. Patients with deep sensory deficit had DM and this finding was associated with diabetic polyneuropathy. Pain was present in the CN dermatome in 11 patients.

In 20 patients, the lesions were seen on MRI examination. Cavernous sinus involvement was observed in 6 and tumour infiltration was observed in 4. One of the patients with cavernous sinus infiltration was diagnosed with Burkitt lymphoma. MRI findings in 3 patients were observed with infectious etiology. Herpes simplex infection was seen in one of these patients, Brucellosis in 1 and tuberculosis meningitis in 1. Brain imaging was within normal limits in 2 patients with Covid-related PCN. One patient with isolated Vth CN involvement on MRI was diagnosed as MOG antibody-associated demyelinating disease.

In 20 patients, CSF protein levels were above 45 mg/dL. One patient had more than 10 leukocytes and was diagnosed with tuberculous meningitis. OCB were type 2 positive in one patient, who was also found to have neurosarcoidosis and a history of recurrent facial paralysis. CSF ACE levels were within normal limits in all patients. Vasculitis markers were positive in 5 patients: anti-nuclear antibody (ANA) was positive in 2 patients, isolated lupus anticoagulant (LAC) in 2 patients, isolated Anti-Neutrophil Cytoplasmic Antibodies (ANCA) in 1 patient, and simultaneous ANA and anti-Scl antibody positivity in 1 patient. Additionally, simultaneous LAC positivity was observed in 2 patients.

Etiological causes were inflammatory causes in 25 patients, ischaemic causes in 23, tumour infiltration in 5 and infectious causes in 6. Among the patients with tumour infiltration, one had a pituitary tumour, one had a clivus metastasis of papillary thyroid cancer and two had leptomeningeal metastases. No parenchymal lesion was observed in any of the patients. In a 70-year-old male patient with a history of pituitary mass removal and IXth PCNP, examinations for the etiology revealed no cause (Table 2). None of the patients were diagnosed with IgG 4-related disease in our study.

Two of the patients with multiple PCNPs had tumour infiltration, two had Herpes infection, two had Covid-19, one had brucellosis and one had tuberculous meningitis. One of the patients with tumour infiltration had clivus metastasis after papillary thyroid cancer and the other had Burkitt lymphoma. Five patients with facial diplegia were diagnosed with GBS. Neurosarcoidosis was found in one patient with recurrent facial paralysis. Ischaemic causes were most commonly related to microvascular damage due to DM. Fifteen patients had VIth, 4 patients had IIIrd, 3 patients had IIIrd+VIth, and one patient had IIIrd+IVth cranial nerve involvement.

IVIg was administered to 5 patients, corticosteroids to 17 patients, and plasmapheresis to 1 patient. Acyclovir treatment was given concurrently with corticosteroid therapy in 1 patient. Among the patients, 14 showed a complete response to treatment, 22 had a partial response, and improvement was observed in 16 patients following blood glucose regulation.

In the 27-month period before the pandemic, 16 patients (0.60 patients/month) and in the 40-month period after the pandemic, 47 patients (1.18 patients/month) were hospitalised due to PCNP. It was observed that no patient with PCNP was hospitalised during the initial period of pandemic when strict restrictions were implemented and the clinical condition of Covid-19 patients was severe. During the pandemic period, the first case was hospitalised on 11 August 2020 (5 months after the first Covid-19 case seen in Türkiye). Similarly, before and after the pandemic, the most common involvement was seen in VIth CN, followed by IIIrd CN and VIIth CN. It was noticed that inflammatory causes were more common in the etiology after the pandemic (Table 3).

Table 1. Distribution of cranial nerve involvements

Cranial nerve/nerves	Count	%
II.	1	1.6
III.	6	9.5
IV.	1	1.6
V.	3	4.8
VI.	24	38.1
VII.	6	9.5
IX.	1	1.6
XII.	1	1.6
III. + IV.	2	3.2
III. + VI.	9	14.3
III. + VII.	1	1.6
VI. +XII.	1	1.6
IV. + VI.	1	1.6
VI. + VII.	1	1.6
II. + III. + V.	1	1.6
III. + IV. + V. + VI.	1	1.6
III. + IV. + VI. + XII.	1	1.6
III. + IV. + V. + VI. + VII.	1	1.6
III. + IV. + V. + VI. + VII. + VIII.	1	1.6

Table 2. Etiologies of cranial neuropathies

Etiology	Number
Inflammatory	25
GBS	5
Demyelinating disease	2
Idiopathic	10
Neurosarcoidosis	1
Tolosa-Hunt Syndrome	7
Ischemic*	23
Infectious	6
Tuberculosis	1
Herpes simplex	2
Covid-19	2
Brucella	1
Malignancy	5
Pituitary	1
Metastasis	4
Colon CA	1
Prostate CA	1
Papillary thyroid CA	1
Burkitt lymphoma	1
Cause undetermined	4

CA: cancer; GBS: Guillain-Barre Syndrome; GBM: Glioblastome Multiforme; MFS: Miller-Fischer Syndrome * Microvascular disease due to diabetes mellitus or hypertension.

Table 3. Impact of the Covid-19 pandemic

		Before the pandemic (N: 16)	After the pandemic (N: 47)
Age (mean ± SD; year)		59.93±11.76	55±16.31
Gender	Woman	56%	43%
	Man	44%	57%
Etiology	Inflammatory	13%	49%
	Ischemic	63%	28%
	Infectious	0%	13%
	Malignant disease	13%	6.4%
The most common PCN	VI. CN	81%	57%
	III. CN	25%	40%
	VII. CN	6%	19%

CN: cranial neuropathy; PCN: peripheral cranial neuropathy; sd: standard deviation.

DISCUSSION

PCNs other than Bell's palsy are rare. Most Bell's palsy or the VIth and IIIrd CN palsies that develop in a patient with known DM for a long time do not require inpatient investigation unless there is no additional feature in neurological examination and cranial imaging. In this study, only 1.75% of the patients who were followed up as inpatients in our clinic, which is a tertiary university neurology clinic, during a 72-month period were found to have PCNP. It was noteworthy that the VIth CN involvement was the most common in the inpatient group. During the Covid-19 pandemic, it was observed that the frequency of patients with CNP with inflammatory etiology examined as inpatients in our clinic, was increased.

The most common CN involvement in outpatient clinics is peripheral facial paralysis. The most common causes of peripheral facial paralysis are primarily Bell's palsy (idiopathic) and secondarily Ramsey-Hunt syndrome (8,9). In our inpatient group, involvement of the VIth CN ranked 3rd. The motor nucleus of the VIth CN is located in the pons, the nucleus related with taste sensation is located in the geniculate ganglion; and the superior salivatory nucleus, which provides parasympathetic efferent fibres, is located in the pontine tegmentum. It innervates all facial mimic muscles and the stapedius muscle, carries the taste sensation of the anterior 2/3 of the tongue, and provides parasympathetic innervation of the salivary glands other than the lacrimal and parotid glands (10). It may be involved in this long route due to head traumas, other infectious and inflammatory causes and tumoral compression.

In our study, the most common CNP was the VIth, seen in 63% of our patients (n: 40), followed by IIIrd CNP in 36.5% (n: 23), VIth CNP in 15.8% (n: 10), and IVth CNP in 6.34% (n: 7). The largest study in the literature on cranial neuropathies is the retrospective study of 979 cases by Keane et al. (11). In this study, the most common involvement was VIth CN with a rate of 57%, followed by VIIth CN with a rate of 47%, and IIIrd CN with a rate of 34% (11). In our group, VIth CN and IIIrd CN were observed with similar rates as in the literature. The fact that Keane et al. included CNPs due to ischaemic stroke in their study explains the fact that CNP of VIth was observed less frequently in our review compared to the literature. In our study, supranuclear or nuclear involvement due to stroke was not included because only peripheral parts of CNs were investigated. In addition, in our group, the most common coexistence of IIIrd and VIth CNs was observed similar to the literature (11).

In our clinic, inflammatory causes were observed in 39.6% (n: 25) and ischaemic causes were observed in 36% (n: 23) of patients with CN involvement who were hospitalised for etiological investigations and treated. The most common inflammatory causes were Tolosa-Hunt syndrome, Guillain-Barre syndrome, demyelinating CNS disorders, and vasculitic processes, and the causes of inflammatory events were similar in the literature (11). Compared to the literature, inflammatory causes were observed more frequently, and tumoral localisation which was 30% in the review by Keane et al. was seen as 7.93% (n: 5) in our study (11). The fact that patients with cranial neuropathy due to tumoral involvement were primarily admitted to neurosurgery department, and that we did not include patients with parenchymal lesions in our study explains the low rate of patients with tumours in our case group.

Tolosa-Hunt syndrome is a rare disorder characterised by painful ophthalmoplegia and idiopathic granulomatous inflammation of the cavernous sinus (12). Clinically, it is characterised by involvement of one or more of the IIIrd, IVth, or VIth CNs, spontaneous recovery and tendency to relapse, unilateral headache, and inflammation in the cavernous sinus, superior orbital sinus or orbit on cranial imaging (13). In our study, Tolosa Hunt syndrome was observed in 32% of CNPs with inflammatory causes. Similar to the literature, the most commonly involved CN was VIth,

followed by CN III and CN IV (13). In a retrospective study by La Mantia et al., in which 124 patients with Tolosa-Hunt syndrome were retrospectively analysed, MRI was found within normal limits in 33% of the patients (14). In our series, 25% had no imaging findings. Corticosteroid treatment was administered to all of our patients and complete response was observed in 58%, partial response was observed in 30% and no response was obtained in only 12%.

Another inflammatory cause is multiple sclerosis (MS), which is a chronic inflammatory, demyelinating and neurodegenerative disease of the central nervous system (15). According to the revised McDonald criteria of 2021, optic neuritis is one of the clinical attacks required to diagnose MS (16). In our series, isolated peripheral cranial neuropathies were examined and optic neuritis patients with MS, Neuromyelitis Optica Spectrum Disorder, Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) were not included in our study. In a study conducted by Zadro et al. that investigates isolated cranial nerve involvement in MS patients, Vth CN was observed to be most frequently involved (17). In our study, one patient with Vth CN involvement was diagnosed with MOGAD. Also two patients with IIIrd CN, one patient with IIIrd and VIth CN involvement diagnosed with MS.

GBS is an acute inflammatory polyradiculoneuropathy and the most common cause of acute flaccid paralysis (18). It is classically defined as a triad of "acute ascending symmetric muscle weakness, areflexia and albumino-cytological dissociation in CSF" (18,19). CN involvement is common in GBS with a rate of 45-70% (20,21). It has been shown that the lower CNs are most commonly involved, followed by the facial nerve. Facial nerve involvement is usually bilateral (20,21). In the diagnostic criteria of facial diplegia, which is one of the rare variants of GBS, the core features are defined as "facial paralysis (may be asymmetric) and areflexia/hyporeflexia (may be normal) in the extremities; absence of ophthalmoplegia, ataxia and limb or neck weakness; reaching to plateau phase of weakness in the period between onset and the following 12 hours to 28 days" (19). The facial diplegia variant, which composed all of the GBS patients in our series, was observed in 20% of all inflammatory etiologies and was observed more frequently than that of the literature (11,22).

CNP due to DM is the most common ischaemic cause, which ranks second in the etiology with a rate of 36.5% (n: 23). Ophthalmoplegia due to IIIrd, VIth and IVth CNs involvement is observed in CNPs due to small vessel vasculopathy of DM (23). In the study by Alzailaie et al., the risk of cranial neuropathy was 10 times higher in patients with DM compared to those without DM (24). In addition, the most common etiological cause was vasculopathy in all groups in the study conducted by Çolpak et al., in Türkiye, in which isolated IIIrd, IVth, and VIth CN involvement was investigated (25). In our series, similar to the literature, ischaemia-induced cranial neuropathies were most commonly seen in IIIrd and VIth CNs. In terms of response to treatment, 22% of our patients had a complete response to treatment, 34% had partial recovery and 25% had spontaneous recovery with blood glucose regulation.

In conclusion, the VIth CN involvement alone/in association with other CNs was the most common type of PCNP. Inflammatory causes (such as Tolosa-Hunt syndrome, neurosarcoidosis, GBS) played the most common role in the etiology of PCNP. Malignancy and infectious etiologies should be considered in patients with multiple CNPs.

Ethics Committee Approval: Approval for this study was obtained from Bezmialem Vakif University Faculty of Medicine Ethics Committee on 26.04.2024 with the number E-54022451-050.04-149093 and written permission was obtained from Bezmialem Vakif University Health Research and Application Centre Bezmialem Vakif University Hospital.

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