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A Study on Etiological and Clinical Profile of Multiple Cranial Nerve Palsies

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Abstract: ***Multiple cranial nerve palsies present a diagnostic challenge due to their diverse etiologies. This observational study aimed to investigate the underlying causes and clinical characteristics of patients with multiple cranial nerve palsies. A comprehensive assessment of 50 patients meeting inclusion criteria was done .Those with known causes of cranial nerve palsies were excluded. Data collected included detailed medical histories, comprehensive physical examinations, routine blood parameters, cerebrospinal fluid analysis, and neuroimaging studies. Among the patients analyzed, 62% were male, with an average age of 42.65 years. Vascular diseases were the predominant etiology, accounting for 36% of cases, Infections were the second most common cause (18%), with tuberculosis and bacterial meningitis identified as significant contributors. Guillain-Barré Syndrome (GBS) and its variants constituted 12% of cases. Tumors (6%) and trauma (6%) were less frequent. Various combinations of cranial nerves were affected, with IX and X cranial nerves being the most frequently involved pair. Multiple cranial nerve palsies pose a complex diagnostic challenge, necessitating tailored clinical judgment for empiric therapy. This study highlights the diverse etiologies associated with multiple cranial nerve palsies useful in the evaluation and management of these challenging clinical presentations.***

Keywords: cranial nerve, vascular, infections, tumors

1. Introduction

The 12 pairs of cranial nerves in nervate most structures of the head and neck. Their afferent and efferent connections traverse the meninges, subarachnoid space, bony structures of the cranium, and superficial soft tissues. Disruption of these nerves canoccur at any site along their course, so that a large number of pathologic processes initially are manifested by cranial-nerve dysfunction[1]. Evaluating the patient with multiple cranial neuropathies presents a unique challenge for the diagnostician. The differential diagnosis is broad and includes many life- threatening processes. A knowledge of the anatomy of the cranial nerves and their involvement in pathologic processes is therefore one of the corner stones of neurologic diagnosis. Just as with any other neurologic presentation, the first step in the evaluation requires correct localization

2. Methods and Approach

To study the etiology and clinical profile of patients with multiple cranial nerve palsies and to establish the associated factors over a period of 15months at Neurology Department Guntur medical college.

Inclusion Criteria: Patients with simultaneous or serial involvement of two or more different cranial nerves

Exclusion Criteria: Myasthenia gravis, Motorneuron disease, post papilledema optic atrophy and surgical causes.

3. Results

Total 50 patients who met the inclusion criteria were analyzed for etiological and clinical profile Out of which 31(62%) were males and 19(38%) females. Mean age was 42.65 years, range 11 to 74yrs.

The most common combinations of involved cranial nerves as a whole were IX ,X cranial nerves(13cases), followed by III, VI (8cases), V, IX, X CNs (6 cases), V, VII, VIII (6 cases), and IX, X, VII (5 cases) .

Table 1: Etiological classification of study group

Etiology	No. of Patients
Vascular Disease	21
Infection	8
Inflamation	3
Gbsand Variants	4
Demylination	4
Trauma	2
Cavernous Sinus Syndrome	5
Tumors	3

Vascular disease was responsible for 21 (42%) of the 50 cases. Infection affects 8 out of 50 (16%), Tumors were responsible for 3(6%) of 50 cases. GBS plus Variants constitutes 4 (8%) out of 50 patients. Out of this 3 are of classical GBS, 1 is of Pharyngo- Cervico Brachial variant. Average age being 46.4 years. Ranges from 26 to 61 years.

The most common combinations of involved cranial nerves as a whole were IX, X cranial nerves (6+5+2=13), followed by III, VI cranial nerves (8 cases), V, IX, XCNs (6cases), V, VII, VIII (6 cases), and IX, X, VII (5 cases) cranial nerves.

In our study brain stem is the most common site (19 cases) of cranial neuropathy. Acute Brain stem Stroke (14 cases) being the most common cause of brainstem involvement.

Table 2: Infectious causes

Infection	No. of patients
TB Meningitis	3
Acute Bacterial Meningitis	2
CSOM	1
Rhino Cerebral Mucormycosis	1
Hansen's Disease	1

4. Discussion

Our study of 50 subjects has been compared to Keane's `(James R . Keane,MD),the largest study to date on Multiple cranial Nerve Palsies with analysis of 979 cases, and various other studies on individual groups of different multiple Cranial nerve palsies. Vascular disease, is the most frequent cause in our study, with 14 cases of stroke (12 ischemic and 2 pontine bleeds,3 ischemic cranial neuropathy (III and VI). Whereas in Keane's study most common cause was tumors followed by vascular. Infection accounted for 9 (18 %) of the 50 cases, whereas in Keane's series, infectious etiologies comprised 10 % of their cases with 48 out of 102 cases secondary to meningitis. Out of 9 cases, 5 are Tuberculous meningitis, 1 Acute Bacterial meningitis, 2 CSOM with Gradenigo's syndrome and one case resulted from Hansen's disease. In our study, tumor etiology comprised about 12 % (6cases). In Keane series tumors were the most common cause of Multiple cranial palsy comprising about 30 % of the cases. Most common tumor in our study is pituitary macro adenoma (3 out of 6), followed by CP angle tumors -- 2 schwannoma and 1 Cp angle meningioma. In our study Pituitary macro adenomas mainly presented with compression over cavernous sinus and presented with cavernous sinus syndrome, involving combinations of III, IV, VI, V CN Combinations. Primary cavernous sinus localizing disorder was in 6 patients. 2 cases were due to TB, 2 were due to Mucor and 2 patients were of Tolosa hunt syndrome. A study on etiology of CSS was conducted by Fernandez S et al where tumors were the most common cause. GBS and its variants, constitutes 5 cases (10%), with mean age of presentation 47.7 years, 4 out of 5 patients with GBS had bulbar involvement. One had Opthalmoplegia. All had facial palsy .Compared to a study of Amita Bhargava et al - 50 % have bulbar and 46% had facial palsy.2 out of 3 ADEM cases presented with bulbar and facial nerve palsy with hemiparesis, demyelination. One is CLIPPERS with bulbarpalsy, ophthalmoplegia and quadriparesis.

5. Conclusion

Literature on cranial neuropathy of multiple etiologies is very limited. Keanes et al reported 979 cases of multiple cranial nerve palsies in the year 2005. Most of the available literature shows multiple cranial neuropathies of single etiologies like head trauma, meningitis etc. In our study vascular diseases followed by infections constituted majority. The patient presenting with multiple cranial nerve palsies remains a formidable challenge to any physician Although many of the causes have specific therapies, the evaluation often leadsto a dead end requiring individual clinical discretion to decide on the best possible empiric therapy.

References

[1] Beal MF. Multiple cranial nerve palsies: a diagnostic challenge . N Engl J Med1990322461-463
[2] Keane JR. Multiple cranial nerve palsies. ArchNeurol 200562:1714-1717
[3] Campbell WW. Brainstem and multiple cranial nerve syndromes. In Campbell WW, ed. Dejong'sThe Neurolhic Examination. 6th ed.Philadelphia: Lippincott Williams and Wilkins; 2005:277-296)
[4] Krupp LB, Banwell B, Tenembaum S. Consensus definitions proposed for pediatric multiple sclerosis and related disorders. Neurology2007;68916suppl2):S7-S12.
[5] Tenembaum S, Chmoles N, Fejerman N. Acute disseminated encephalomyelitis: along-term follow-up study of84 pediatric patients. Neurology 2002; 59(8): 1224-1231.