

Full Length Research Paper

Cranial neuropathies in El-shaab teaching hospital (Sudan)

Abbasher Hussein, Bedraldin Mubark, Amira Sidig, Abdla A. Rahman, Ahmad Babikir, Faroug Yassien, Ahmed Hamad, Omer el-Adil, Ahmad Saad, Mohmad Malk Aldar, Esam Mahmoud Ahmad

El-shaab Teaching Hospital, Sudan

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Cranial nerves palsies are one of the most important manifestations of neurological diseases. The aim of the work was to study the etiologies and pattern of presentations of cranial nerves palsies among adult Sudanese patients seen at El-shaab Teaching Hospital in the period from July 200-February 2009. 667 adult Sudanese patients with neurological diseases were included in the study. A full detailed history, proper clinical examination and required investigations including CT scan and MRI of the brain were carried out. The results showed that cranial nerves VII (197 cases), II (121 cases), IX (65 cases), X (65 cases), VI (39) were the most commonly affected, while III (13cases), XI (8 cases) and IV (2cases) were the least affected. Chief causes were stroke (144 cases), space occupying lesions (57 cases), idiopathic intracranial hypertension (25 cases), Guillain Barre syndrome (22 cases), diabetes mellitus (20 cases), hypertension (19 cases), motor neuron disease (19 cases), Bell's palsy (13 cases), multiple sclerosis (12 cases), vasculitis (10 cases), meningitis (7 cases), acquired immunodeficiency syndrome (6 cases), anti phospholipids antibody syndrome in four patients, sensory neural deafness in four cases and trigeminal neuralgia in 4 cases. In addition, other rare causes were observed, such as hydrocephalus in 3 patients, road traffic accident in 2 patients, one patient had retinitis-pigmentosa, one patient had Dandy Walker syndrome and one patient had Vogt Koyangi Harada syndrome. The study showed that there was a wide variety of diseases and locations of cranial nerves involvement. The seventh and the second were the most common cranial nerves involved. Stroke and space occupying lesion were the most common causes of cranial neuropathies.

Key Word: Cranial neuropathies, Sudanese Patients

INTRODUCTION

Cranial nerves are composed of twelve pairs of nerves that emanate from the nervous tissues of the brain. In order to reach their targets, they must ultimately leave the cranium through openings in the skull, hence their name is derived from their association with the cranium (Juncos-Labia, 1987). The motor components of the cranial nerves are derived from the cells that are located in the brain. These cells send their axons out of the cranium where they will ultimately control eye muscles, glandular tissue and specialized muscles. The sensory component of cranial nerves originates from collection of cells that are located outside the brain. In general sensory ganglia of the cranial nerves send out branch that divided into two branches,

a branch that enters the brain and one that is connected to sensory organs. The cranial nerves (with the exception of the first and the second cranial nerves) originate in the brainstem, which includes the midbrain, the pons, and the medulla. The twelve cranial nerves can be divided into sensory, motor, or mixed nerves (Keane 1986). Overall, sensory nerve nuclei tend to be located in the lateral brainstem, while motor nuclei tend to be located medially. Nerves with mixed sensory and motor fibers must have more than one nucleus of origin, at least one sensory (afferent) and one motor (efferent). Sometimes more than one nerve originates from a single nucleus; for example, the sense of taste is spread across at least two nerves, but merges into a single nucleus (Symonds 1958). Any sensory nucleus is receiving input from the periphery, but the sensory receptor cell bodies are never in the nucleus itself. They will always be located just outside the central

*Corresponding author Email: abbashar59@yahoo.com

nervous system in a ganglion (Klin 1982 and Masson et al 1983).

Cranial nerves may be affected singly or in groups and knowledge of which nerves are involved helps locate the lesion. Injuries of the cranial nerves (CN) are relatively common, especially secondary to head trauma. Injuries may be direct or indirect, through damage caused by shear, distraction, or compression. Upper cervical lesions can involve the lower CN IX–XII, because the 1st and 2nd cervical vertebrae are in anatomical proximity to the base of the skull and to CN VI, whose intracranial course is precariously long. Sekhar and colleagues reported a high success rate for reconstruction of CN III–VI traumatized during surgery (Sekhar et al, 1992).

Non traumatic lesions can be degenerative, infectious, or vascular (sudden onset), but most frequently they are secondary to tumors (primary or metastatic). Some of the causes of cranial nerve lesions are given below. Conditions which can affect any cranial nerve include: (1) Diabetes mellitus (2) MS (3) Tumours (primary or secondary) (4) Sarcoid (5) Vasculitis (e.g. polyarteritis nodosa) (6) Systemic lupus erythematosus (7) Syphilis (8) Chronic meningitis (9) Guillian Barre Syndrome (10) Lyme disease. (11) Stroke (12) HIV.

Ransom and associates reported a series of patients with prostate cancer and metastases to the base of the skull who presented with symptoms and signs of different cranial nerve lesions (Ransom et al, 1990). The exact incidence of cranial neuropathies in our country is not known.

Objective

The objective of this study was the description of etiologies and pattern of clinical presentations of cranial neuropathies among adult Sudanese patients seen at El-shaab Teaching Hospital, in the period from July 2007 to February 2009.

MATERIALS AND METHODS

This is a descriptive study based on the analyses of brain cross sections conducted at El-shaab Teaching Hospital (Sudan). El-shaab Teaching Hospital is a tertiary hospital with 243 beds, located in the centre of Khartoum. The study population included 667 patients with neurological diseases admitted or referred to El-shaab teaching hospitals in the period from June 2005 to February 2006. All patients were Sudanese that gave their verbal consent to participate in the study, those below 18 years were excluded. A full detailed history was taken from each patient and systemic examination was performed by the authors. The history included symptoms in favour of cranial nerves involvement like loss of smell, visual impairment, diplopia, facial pain and numbness, deviation of the mouth, accumulation of saliva, hearing defect, vertigo, dysphagia, inability to protrude the tongue, headache, convulsions, unsteadiness, dizziness, loss of consciousness, upper and lower limbs weakness, numbness and sphincteric disturbances. The physical signs were

grouped into general, systemic and neurological categories. The following investigations were done if needed: Urine general, stool general, Chest X-ray, Mantoux test, Sputum for Acid and Alcohol fast bacilli, complete haemogram, CT brain, MRI brain or spinal, CSF studies, Nerve conduction studies, EMG, HIV serology and investigations looking for evidence of vasculitis like antiphospholipids antibodies. **Data analysis:** The data was analyzed using computer program the Statistical Package for Social Sciences System (SPSS-11), Chi-square test, student t-test were used and a P value of less than 0.05 was considered statistically significant.

RESULTS

The study showed that out of 667 patients, 419 had cranial neuropathies, 52.6 % were females. Age distribution was found as follows: 43 patients between 18–20 years, 93 patients between 21–30 years, 73 patients between 31–40 years, 58 patients between 41–50 years, 41 patients between 51–60 years, 59 patients between 61–70 years, 39 patients between 71–80 years, 9 patients between 81–90 years while only two patients between 91–100 years. Regarding the onset of the disease, in 42% it was of sudden onset; in 9 % it was of rapid onset while in 49% it was of gradual onset. Table 1 shows distribution of cranial nerves involvement in 419 Sudanese patients with cranial neuropathies.

It did appear that 8 patients presented with cranial neuropathies due to rare causes such as hydrocephalus in 3 patients, 2 had road traffic accident, one had retinitis-pigmentosa, one had Dandy Walker syndrome and one patient had Vogt Koyangi Harada syndrome (table 2).

Clinical examination of the third, fourth and sixth cranial nerves showed that 66 patients had diplopia while 52 presented with ptosis, 10 had right oculomotor nerve palsy, 3 had left oculomotor nerve palsy, one had right trochlear nerve palsy, one had left trochlear nerve palsy, 9 had right sixth nerve palsy, 14 had left sixth nerve palsy in addition to 16 patients with bilateral sixth nerve palsy. The pattern of clinical presentation of the fifth cranial nerve was as follows: 22 patients had difficulties of mastication, 4 complained of pain during chewing, and 4 patients had hyperesthesia (table 3). Examination of the facial nerve showed that 197 patients complained of mouth deviations, dripping of saliva, accumulation of fluid and loss of taste, 51 patients were unable to close their eyes, while 12 patients had hyperacusis. It was found that 93 patients had UMN facial palsy, 15 had LMN facial palsy, 53 had left UMN facial nerve palsy, 13 had left LMN facial nerve palsy in addition to 23 patients with bilateral LMN facial palsy. The study revealed that 15 patients had difficulties of hearing, 6 had vertigo, 2 had right sided hearing impairment, 4 had left sided hearing impairment while 9 patients had bilateral impairment. It appeared that 65 patients had dysphagia, 36 had right ninth and tenth cranial nerves palsy and 17 had left ninth and tenth cranial nerves palsy while 12 patients had bilateral affections. Examination of the accessory nerve showed that 8 patients complained of failure to

Table 1. Distribution of cranial nerves involvement in 419 Sudanese patients with cranial neuropathies

The cranial nerve	Number	Percent
First cranial nerve	3	00.52%
Second cranial nerve	121	21.15%
Third cranial nerve	13	02.27%
Fourth cranial nerve	2	00.35%
Fifth cranial nerve	22	03.84%
Sixth cranial nerve	39	06.82%
Seventh cranial nerve	197	34.44%
Eighth cranial nerve	15	02.62%
Ninth cranial nerve	65	11.36%
Tenth cranial nerve	65	11.36%
Eleventh cranial nerve	8	01.40%
Twelfth cranial nerve	22	03.84%
Total	572	100%

Table 2. Shows the causes of cranial neuropathies among our studied group.

Causes of the cranial nerves palsy	frequency	percent
Stroke	144	34.40%
Space occupying lesions	57	13.60%
Myasthenia gravis	42	10.00%
Benign intracranial hypertension	25	06.00%
Gillian Barri syndrome	22	05.30%
Diabetes millutes	20	04.80%
Motor neuron disease	19	04.50%
Hypertension	19	04.50%
Bell's palsy	13	03.10%
Multiple sclerosis	12	02.90%
Vasculitis	10	02.30%
Meningitis	07	01.70%
Human immunodeficiency syndrome	06	01.40%
Trigeminal neuralgia	04	00.90%
Anti phospholipids syndrome	04	00.90%
Idiopathic sensory neural deafness	04	00.90%
Common cold	03	00.71%
Rare causes	8	01.90%
Total	419	100%

shrug their shoulder, one had right eleventh cranial nerve palsy, 2 patients had left eleventh cranial nerve palsy in addition to 5 patients who had bilateral affection. The pattern of clinical presentation of hypoglossal nerve palsy was found as follows: 19 patients had twelfth cranial nerve palsy who complained of either failure to move the tongue, had tongue deviations or had fasciculation's and wasting of the tongue. Table 4 shows the causes of space occupying lesions among our studied group, while table 5 shows CT brain findings with stroke patients.

DISCUSSION

Cranial nerve involvement had no gender predilections 40 (Wong 1997 and Trevino 1995). The age group between

-70 years was the commonly involved , this is due to the increased number of incidence of stroke and brain tumors among this age group, similarly to that mentioned by Juncos Labia, 1987. The variation in the onset of symptoms of cranial neuropathies was found to be from gradual onset in 49.5% of the cases to sudden in 42% and rapid in 9% which was due to different etiologies. The symptomatology of brain tumors was of gradual onset while stroke symptoms were either sudden or rapid. Other diseases like benign intracranial hypertension, Guillain Barre syndrome, motor neuron disease, multiple sclerosis and meningitis showed symptoms of gradual onset, similar result was reported by Kodsí and Younge 1992. Cranial nerves seventh, second, ninth and tenth were the commonly affected nerves, the locations and causes were

Table 3. Shows fundal changes among our studied group

Fundal	Changes	Number	100%
	Bilateral papilloedema	37	5.50%
	Bilateral optic atrophy	33	4.90%
	Hypertensive retinopathy grade I-III1	19	2.80%
	Diabetic retinopathy	19	2.80%
	Bitemporal hemianopia	05	0.74%
	Bilateral corioretinitis	03	0.40%
	Bilateral papillitis	03	0.40%
	Retinitis -pigmentosa	01	0.14%
	Bilateral retinitis	01	0.14%

Table 4. Shows the causes of space occupying lesions among our studied group

Causes of space occupying lesions	frequency	percent
Meningioma	16	28.10%
Sub dural haematoma	09	15.80%
Pituitary adenoma	08	14.00%
Cerebellopontine angle tumor	07	12.30%
Sagittal sinus thrombosis	05	08.80%
Astrocytoma	04	07.00%
Brain metastasis	02	03.50%
Tuberculoma	02	03.50%
Haemangeoma	01	01.70%
Cerebral toxoplasmosis	01	01.70%
Brain abscess	01	01.70%
Occipital proencephalic cyst	01	01.70%

Table 5. shows CT brain findings among those who had stroke

Imaging findings	Number	Percent
Left cerebral infarct	40	27.80%
Right cerebral infarct	35	24.30%
Brain stem infarct	11	07.60%
Bilateral infarct	06	04.20%
Multiple infarct	03	02.10%
Peri ventricular infarct	01	00.70%
Normal finding	24	16.70%
Right cerebral hemorrhage	12	08.30%
Left cerebral hemorrhage	12	08.30%

diverse. The incidence of seventh cranial nerve palsy was increased because it was associated with clinical presentations of cerebrovascular accident. Second cranial nerve involvement is one of the most important clinical manifestations of brain tumors, benign intracranial hypertension, diabetes mellitus, hypertension and multiple sclerosis, similar to the result obtained by Symonds CP 1958. Similar to what was mentioned in the literature the study showed that ninth and tenth cranial nerves palsies were commonly seen with stroke, motor neuron disease and Guillain Barre syndrome (Daniell et al, 1996). Out of 144 patients with stroke, cerebral infarction was seen in 120, while hemorrhage occurred in 24 patients. The space

occupying lesions were detected in 57 patients (13.6%), these lesions included meningioma in 16 patients (28.1%), subdural hematoma in 9 patients (15.8%), pituitary adenoma in 8 patients (14%), cerebellopontine angle tumors in 7 patients (12.3%), sinus thrombosis in 5 patients (8.8%), astrocytoma in 4 patients (7%), brain metastasis in 2 patients (3.5%), tuberculoma in 2 patients (3.5%), hemangioma in one patient (1.7%), cerebral toxoplasmosis in one patient (1.7%), brain abscess in one patient (1.7%), and occipital proencephalic cyst in one patient (1.7%). Other causes of cranial neuropathies included myasthenia gravis in 42 patients (10%), benign intracranial hypertension in 25 cases (6%), Gillian Barre

syndrome in 22 patients (5.3%), diabetes mellitus in 20 patients (4.8%), motor neuron disease in 19 patients (4.5%), hypertension in 19 cases (4.5%), Bell's palsy in 13 patients (3.1%), multiple sclerosis in 12 patients (2.9%), vasculitis in 10 patients (2.3%), meningitis in 7 patients (1.7%), human immunodeficiency syndrome in 6 patients (1.4%), trigeminal neuralgia in 4 patients (0.9%), antiphospholipids antibody syndrome in 4 patients (0.9%), idiopathic sensorineural deafness in 4 patients (0.9%), common cold in 3 patients (0.71%). In other 8 patients (1.9%) cranial neuropathies were due to rare causes like hydrocephalus in 3 patients, road traffic accident in 2 patients. This result was different from that described by Kaene where road traffic accident was found to be the commonest cause of cranial nerves palsies (Kaene 1996). Regarding the localization of the cranial nerve involvement the commonest causes were stroke, space occupying lesions and sinus thrombosis. This result was similar to what was mentioned in the literature earlier¹³. Brain stem involvement occurred in 11 patients, the non localizing involvement dominated by Gillian Barre syndrome, Bell's palsy, motor neuron disease, benign intracranial hypertension, diabetes mellitus and hypertension, similarly to that reported by Juncos Labia MF. No patient had involvement of all cranial nerves, although some patients had involvement of more than six cranial nerves unilaterally, similarly to that reported by Kaene 1996. loss of smell can occur in some neurological diseases, but our study showed that only 3 patients (0.71%) had bilateral loss of smell due to recurrent common cold (Trobe 1985 and 1988). Bilateral papilloedema and bilateral optic atrophy were found to be the commonest fundal changes representing the superiority of space occupying lesions as one of the commonest causes of cranial neuropathies. Diabetic and hypertensive retinopathy indicated non localizing causes of multiple cranial nerves palsies. Diplopia was a symptom that indicated involvement of oculomotor, trochlear and abducent nerves (Acierno et al, 1995, Brazis 1985 and Lang 1985). Involvement of the sixth cranial nerve was often a false localizing signal, although the incidence of the sixth cranial nerve palsy in our study was different from other studies, most likely it was due to the small sample size in our study (Dubner 1987). The pattern of involvement of the fifth cranial nerve was similar to what was mentioned in the literature Eide and Rabben 1998). Out of the 197 patients (47.02%) presented with facial palsy, 146 had UMN lesion. Stroke is the commonest cause of cranial neuropathies, therefore UMN lesion of the facial nerve was a well known recognized clinical feature of CVA (Jacobson et al, 1997). Only 11 patients had facial palsy in the opposite side of the affected limbs representing brain stem infarctions (cross hemiplegia). The number of patients with LMN lesions facial palsy were 41 (12.19%), they were affected by Bell's palsy and GBS in addition to Ramsay Hunt syndrome, leprosy and sarcoidosis which were very rare causes (Dickins 1988). Hyperacusis and loss of taste

were seen in 12 patients (2.86%) with Bell's palsy and were considered as less useful prognostic features (Addington et al, 1999). Vertigo was observed in 16 patients (3.81%) and difficulties of hearing was detected in 15 patients (3.58%), which symptoms were the most frequent ones of the eighth cranial nerve palsy, that mainly affected bilaterally. Dysphagia was the commonest symptoms in case of ninth and tenth cranial nerves palsies, stroke and motor neuron disease were found to be the most frequent causes. The eleventh cranial nerve involvement was either idiopathic, part of manifestations of stroke or tumors involving brain stem. Twenty two patients presented with hypoglossal nerve palsy motor neuron disease and stroke where the most important causes, similarly to the result obtained by Keane.

CONCLUSION

There was a wide variety of diseases and locations of cranial nerves involvement. The seventh and the second were the most common cranial nerves involved. Stroke and space occupying lesion caused cranial neuropathies most frequently.

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