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Post-Traumatic Complete Paralysis of the III Pair of Cranial Nerves at Sikasso Hospital: A Case Report

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Abstract | **Case Report**

Public road accidents (AVP) are frequent and serious and are responsible for numerous cranioencephalic and neurological lesions. Paralyzes of the oculomotor nerves are frequently reported, especially in young subjects during these AVPs. We report the case of complete paralysis of the third pair of cranial nerves in a 22-year-old young lady, following a road accident. Clinical examination revealed intrinsic and extrinsic paralysis of the 3rd pair of cranial nerves. Computed tomography showed a subarachnoid hemorrhage. After 7 days of hospitalization under analgesics and hyperhydration, a complete regression of the lesions was obtained. An angio-scan performed 3 months after the accident showed a slight non-significant stenosis of 25. 54% of the left internal carotid without anomaly of the intracranial vascular network. Paralyzes of the third cranial pair, total or partial, most often regress within variable periods of 6 months to 2 years. The prevention of accidents on the public highway requires the application of road safety measures.

Keywords: Sub arachnoid haemorrhage, III nerve palsy, AVP.

INTRODUCTION

In 2018, according to the WHO, on global road safety, YLL-related deaths reached 1.35 million in 2016, injured 25-30 million and continues to rise steadily as death rates from relative to the size of the world population remain constant [1]. Paralysis of the ocular motor cranial nerve is a frequent condition in ophthalmology departments throughout the world and is a major cause of visual morbidity [2]. The third cranial pair is very important because it participates in the extrinsic innervation of 5 muscles: the internal rectus, the superior rectus, the inferior rectus, the inferior oblique, and the levator of the superior eyelid. It also intervenes in the intrinsic innervation of the sphincter of the pupil and the ciliary muscle by its parasympathetic fibers. This complexity of innervation explains the clinical polymorphism of III paralysis [3].

Microvascular ischemia is the leading cause (39.4%), followed by trauma (30.3%), aneurysms (15.2%), tumors (9.1%) and undetermined causes (6.1 %) [2].

We report the case of a complete paralysis of the III pair of cranial nerves in a young lady of 22 years, following an accident on the public road between motorbike and we then discuss the modalities of the therapeutic management of ptosis linked to the paralysis of the III cranial pair.

OBSERVATION

We report the observation of a 22-year-old student admitted to the emergency department for cranial trauma with an initial loss of consciousness of 2 hours following a public road accident between motorcycle. She complained of headaches and a falling of the eyelid of the right eye. Clinical examination revealed a Glasgow score of 15/15, ptosis (fig. 1) and scratches on the body. There was no meningeal sign or motor deficit in all four limbs..The ophthalmological examination found uncorrected visual acuity of 3/10 in the right eye with major ptosis associated with limitation of movements inside, below and above, associated with mydriasis. Intraocular pressure was 20 mmHg and fundus was normal without Terson's syndrome with a cup to disc ratio of 0.2. The left eye had an uncorrected visual acuity of 8/10 with a history of optical correction not worn at the time of the accident. The remainder of the examination was unremarkable. A cerebral computed tomography performed on admission had shown a subarachnoid hemorrhage (fig. 2) leading to hospitalization and hyperhydration and analgesic treatment with paracetamol 1g every 8 hours in hospital for 7 days.

Figure 1: Major ptosis OD

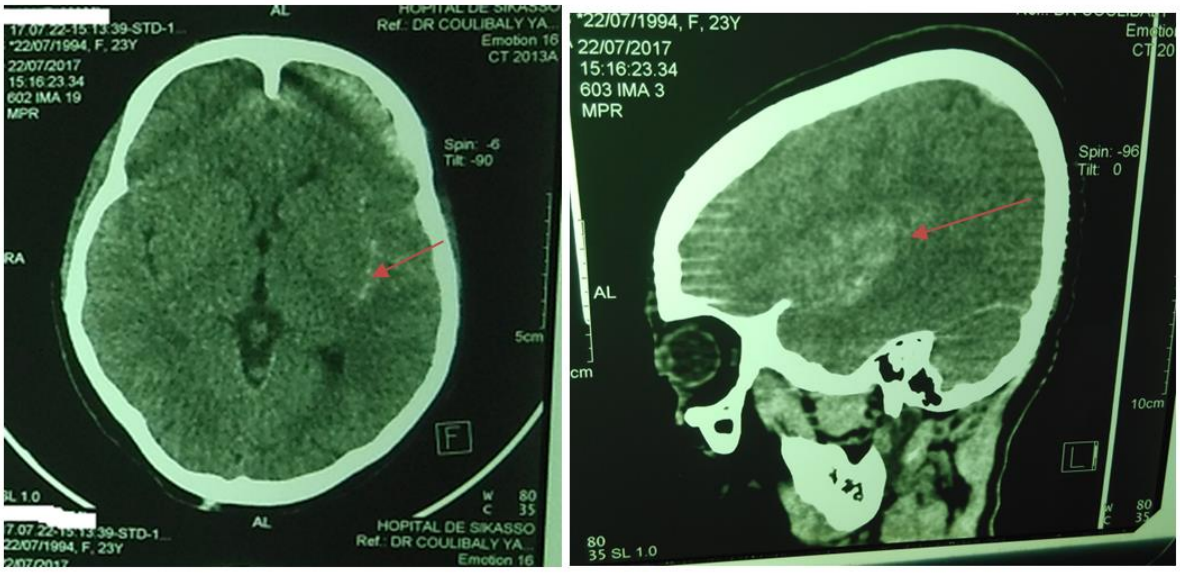

Figure 2: Axial and sagittal section: Spontaneous hyperdensity at the level of the left sylvian valley (subarachnoid hemorrhage)

At 3 Months of Evolution

We observed a regression of ptosis (fig. 3) and an increase in visual acuity to 5/10. The regression was total and without anatomical or functional sequelae.

An angio-scan performed 3 months after the accident did not show any malformation of the intracranial vascular network, on the other hand a slight non-significant stenosis of 25.54% of the left internal carotid was noted (fig. 4).

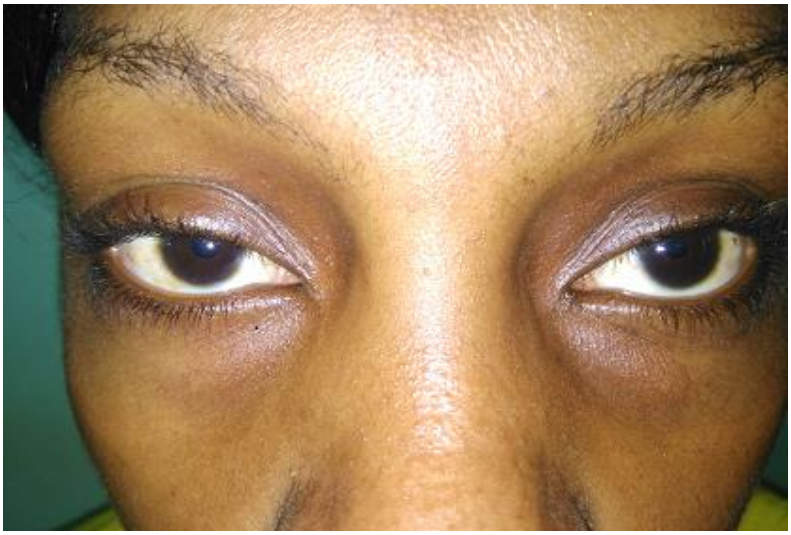

Figure 3: Regression of ptosis

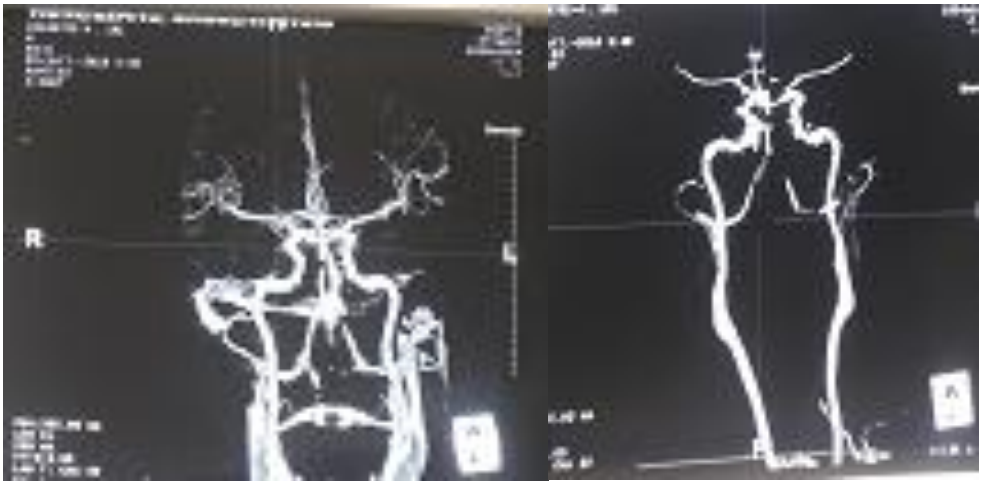

Figure 4: CT-ANGIO CEREBRAL non-significant stenosis of 25.54% of the left internal carotid

DISCUSSION

Paralysis of the III rd cranial pair is an attack whoseincidence is 3.5 to 4 cases per 100,000 population in adults. This incidence would be greater in the over 60s compared to the under 60s [2].

A complete III palsy therefore leads to a divergent strabismus with a vertical element and a ptosis associated with a deficit of elevation, depression, adduction and mydriasis. But the attack can also be incomplete with absence of mydriasis (pure extrinsic attack) or partial oculomotor deficit. Due to the superior and lateral location of the pupillary fibers, mydriasis may be the first sign of III compression (aneurysm at the posterior communicating junction-internal carotid or pituitary apoplexy [4].

In general, the main etiology in adults remains dominated by microvascular damage with 35 to 40% of cases, followed by trauma, compression due to a tumor and sequelae of neurosurgery with respectively12%,11% and 10% [5, 6].

M. Nurul-Ain *et al.*, [2], in Malaysia, showed that the most common cause of isolated third cranial nerve palsy was microvascular ischemia (39.4%), followed by trauma (30.3%), aneurysms (15.2%), tumors (9.1%) and undetermined causes (6.1%).

Our 22-year-old patient had an attack of traumatic origin, which fits well with the data of the literature or the traumatic causes of acquired paralysis of the III pair is the most frequent between childhood and early adulthood [6].

The traumatic cause which would represent between 12 to 19% is evoked in the context of serious head trauma [5], which was the case of our patient who had suffered a severe trauma with an initial loss of consciousness.

Pupillary involvement a type of mydriasis was present in our patient. This pupillary attack is written in the literature during the paralysis of the III [6]. This pupillary involvement was observed in 43% of patients during the consultation, but 86% of patients presented

with ptosis at the first visit [6]. Ptosis was found in our patient where it wasassociated with a deficit of elevation, depression, adduction and mydriasis, leading to the diagnosis of III palsy.

By gender, the majority of studies found no significant difference in the incidence of third cranial nerve palsy [6-13]. However, it is interesting to note that some research reports a higher incidence rate in men (2.5 to 3 times higher than in women); especially in the younger age group [14-16].

Our patient had unilateral involvement, which is comparable to that reported by M. Nurul-Ain *et al.*, [2], or all patients had unilateral paralysis. According to the authors, bilateral involvement is rare and most often occurs in less than 10% of patients with paralysis, with various etiologies; including midbrain cavernous malformation, pituitary apoplexy, and giant cell arteritis [17, 18].

Complete III palsy manifests as divergent strabismus with a vertical element and ptosis associated with elevation, depression, adduction deficit and mydriasis [4]. These data from the literature of clinical manifestations agree with the clinical characteristics presented by our patient.

In post-traumatic injuries, eye pain or headaches have been reported in the literature [5]. Which is comparable to our report.

The place of imaging in the etiological and therapeutic diagnosis in III palsy is very important. According to the literature, cerebral computed tomography can be used as the main tool for neuroimaging in cases of III palsy; especially for people with headaches, periorbital pain, anisocoria, trauma, infectious, inflammatory and infiltrative cases [2].

Cerebral computed tomography (CT) revealed a subarachnoid hemorrhage in our patient.

MRI with contrast is superior to CT in terms of soft tissue resolution, for detailed evaluation of tumours, tumours, infectious, inflammatory and infiltrative cases [2]. For lack of a technical platform, we were unable to perform the MRI.

The management consisted of a 7-day hospitalization with hyperhydration associated with the prescription of analgesics at standard doses. After 3 months of follow-up and observation, a significant regression of symptoms was observed with an increase in visual acuity to 5/10. The regression was total and without anatomical or functional sequelae. An angio-scan performed 3 months after the accident did not show any malformation of the intracranial vascular network, on the other hand a slight non-significant stenosis of 25.54% of the left internal carotid was noted.

Paralyzes of the third cranial pair, total or partial, most often regress within variable periods of 6 months to 2 years. After this period, the problem of functional and aesthetic rehabilitation arises [19]. In our case, we observed a regression of damage to the third cranial pair after 3 months of observation.

CONCLUSION

Complete paralysis of the 3rd pair of cranial nerves can completely regress after three months without functional or aesthetic sequelae. The prevention of accidents on the public highway requires the application of road safety measures.

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