# Third cranial nerve paresis in the Emergency Department – reviewing a clinical case

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**Abstract**

Third cranial nerve paresis, although uncommon, is a clinical situation with a broad differential diagnosis which may be associated with severe disease, particularly intracranial aneurysms, with high morbidity and mortality.

The authors present the case of a 40-years-old woman admitted at the Emergency Department with headache, diplopia and right palpebral ptosis for the last fortnight. Furthermore, the patient mentioned impaired coordination of voluntary movements for the last year, with an acute confusional episode about 2 months earlier. Regarding her personal history she was a smoker, with light alcohol habits and slight tricuspid insufficiency, She had been submitted to hysterectomy for cervical dysplasia. There was a history of recent right acute otitis media, as well as a homolateral dental abscess.

On physical examination, there was right palpebral ptosis, light anisocoria, binocular diplopia, abolished right direct and consensual photomotor reflexes, adiadochokinesis and light dysmetria in the finger-nose test, particularly with the left superior extremity.

As the head CT scan was normal, the patient was admitted to the Internal Medicine Department in order to clarify the clinical frame.

On the 3rd day of admission, she mentioned worsening of the headache, another CE CT-scan was carried out, with intravenous contrast showing subarachnoid hemorrhage. 3D reconstruction displayed an aneurysm on the right posterior communicating artery.

The authors discuss the diagnostic approach used in this particular case, as well as the correct approach (according to literature) in a situation of third cranial nerve paresis.

Key words: third cranial nerve paresis, intracranial aneurysm, posterior communicating artery.

## INTRODUCTION

Third cranial nerve paresis is an uncommon clinical situation1 in the daily clinical practice of an Internist.

It may occur in isolation, or in association with other neurological alterations, which helps the localization of the lesion, which may occur at any level of the trajectory of the oculomotor nerve or the respective nucleus.[2]

Third cranial nerve paresis is classified as complete or incomplete, and very importantly for the prognosis, pupil-sparing or not.[1]

The most common etiology is ischemia. However, the most feared is compression by expanding intracranial aneurysm, which in the majority of cases, is not identifiable in CT-CE without contrast.[3]

The differential diagnosis, particularly when the paresis is incomplete or in an initial phase, may be extensive, which is due mainly to the wide spectrum of possible clinical presentations.[1]

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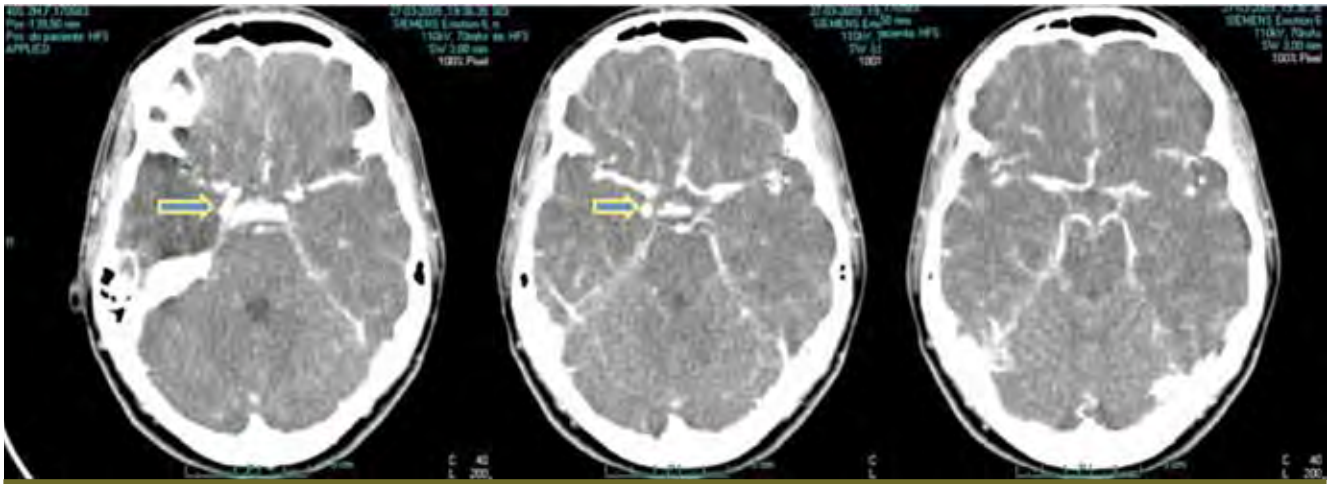
## CASE REPORT

Female patient, aged 40 years, with leukoderma, who visited the Emergency Service due to progressive worsening of headaches, diplopia and right palpebral ptosis with 2 weeks of evolution.

She was asymptomatic until around one year previously, when she had difficulty coordinating voluntary movements, particularly the fine motricity ("difficulty holding small objects"). Two months previously, she reported an acute confusional episode without any apparent cause. Two weeks before coming to the Emergency Service, she developed onset of right frontal migraine of the "heavy type", with average intensity, intermittent, sometimes spreading to the parietal and supraciliary regions on the same side. She also reported, simultaneously, "difficulty fully opening the right eye" and diplopia in the eye "upwards, downwards, and to the left". Four days previously, she mentioned general malaise and headache, which became more intense and holocranial. Various family members and friends said "the right eyelid is drooping". On the previous day, the ptosis of the right eyelid became more accentuated and the diplopia in the above mentioned positions became more marked.

In terms of personal history, there was a history of



CT-CE images with IV contrast carried out no the 3rd day of hospitalization. The arrows show slight prepontic subarachnoid hemorrhage of the suprasellar cistern and proximal portion of the Sylvian fissures on the right, and an apparent saccular aneurysm of the right posterior communicating artery, subsequently confirmed by 3D reformatting.

**FIG. 1**

Pelvic Inflammatory Disease at the age of 31, G3P2 by spontaneous abortion, a total hysterectomy at 37 years by dysplasia of the cervix, and excision, during the same surgery, of an "ovarian cyst", a slight tricuspid insufficiency detected 3 years previously in echocardiogram of the Occupational Medicine, a bilateral breast implant in the previous year for esthetic reasons, a smoker (24 pack-years), drinks around 30 to 40g alcohol/day, previous use of oral contraceptives (from 18 to 26 years). Three weeks before coming to the Emergency Service, there was a history of acute otitis media on the right and around 1 week afterwards, she was medicated with antibiotic (she could not say which one) for a tooth abscess on the same side. In terms of professional activity, the patient reported that she gave home support during the day, and worked in a bar at night. She had a history of unprotected sex with more than one partner.

There was no other relevant personal or family history.

On objective examination, cardiac auscultation revealed a grade II/VI systolic murmur in the tricuspid focus, without irradiation, increasing in intensity when breathing in, and neurological exam revealed ptosis of the right eyelid, slight anisocoria, diplopia on looking upwards, downwards and to the left, direct and consensual photomotor reflexes abolished on the right, adiadochokinesia and slight dysmetria in the finger-to-nose test, which was more evident with the left arm.

The patient was sent to the Ophthalmology Emergency department, which confirmed incomplete paresis of the third right cranial nerve with apparent pupil involvement, manifested as slight anisocoria, with enlarged pupil diameter on the same side. This was also observed by the Neurology department, which suggested a CT-CE and careful monitoring. The patient subsequently deteriorated while in the care of Internal Medicine.

CT-CE performed on entry to the Emergency Service, without intravenous contrast and axial sections of 4mm in thickness, did not reveal any type of alterations.

The patient was admitted to the Internal Medicine Service for monitoring and clarification of the clinical situation.

From the laboratory exams requested, the following results are highlighted: erythrocytes $3.48x10^{12}$/L, hemoglobin 11.5 g/dL, hematocrit 32.7 % , MCH 33 pg, prothrombin rate 80.4%, INR 1.21, fibrinogen 476.3 mg/dL, ESR 50 mm, CRP 7 mg/L, C3 81 mg/dL, IgG anti-rubella 180 UI/ml, IgG anti-toxoplasma59.1 UI/ml, EBNA G 99.2 e VCA G 659, with respective IgM within the normal limits.

The remaining hemogram, coagulation, fasting glycaemia, renal, hepatic and thyroid function, serum ionogram, uricaemia, serum protein electrophoresis, total proteins and albumin, muscle enzymes, lipid

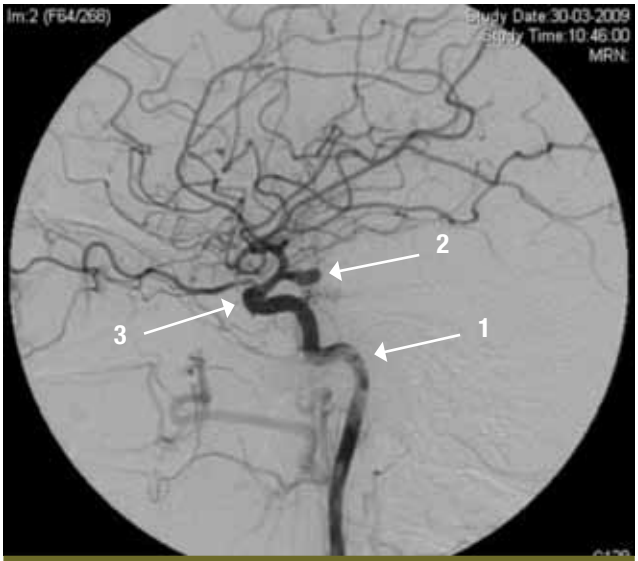



Image of the angiography carried out on the 3rd day post-rupture of the aneurysm, showing the inner carotid (1) and the sacular aneurysm of the right posterior communicating artery (2) of around 8x5mm. Though less evident, a small aneurysm of the carotid cavum on the same side is also evident (3)

**FIG. 2**

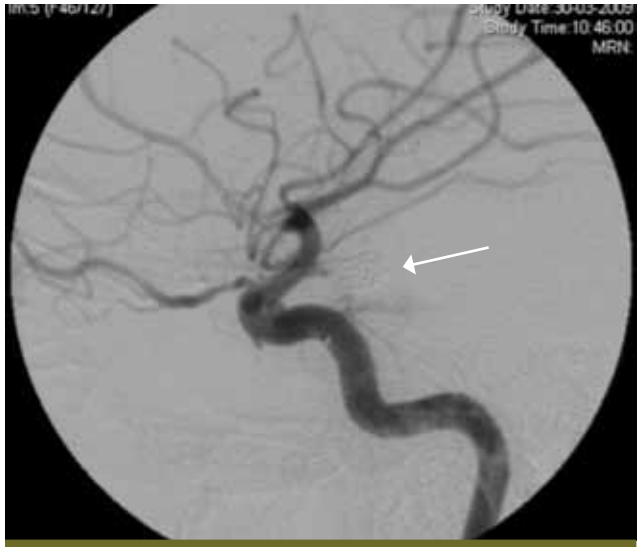



Image of the angiograph performed post-embolization of the aneurysm of the right posterior communicating artery.

**FIG. 3**

profile, vitamin B12 and folic acid did not show any alterations. The tumor markers, antibodies and syphilis and viral serologies were negative.

The summary urine analysis revealed only some leukocytes, some erythrocytes and a high number of epithelial cells.

The ECG and anteroposterior teleradiography of the thorax did not reveal any relevant alterations.

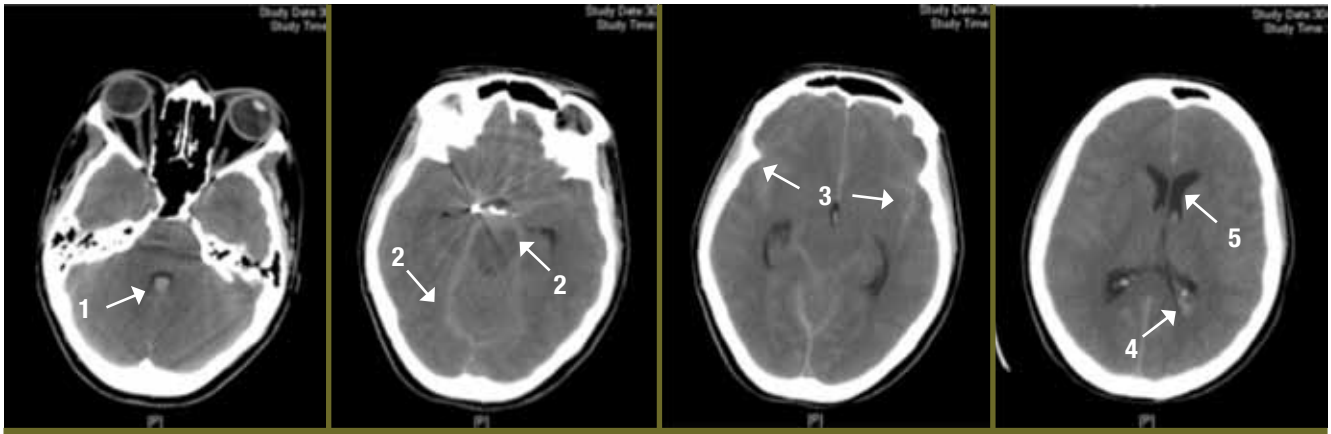
On the 2nd day of hospitalization, lumbar puncture was performed, with normal opening and exit pressure of transparent liquid, of the "rock crystal" type. The LCR exam revealed six nucleated cellular elements/mm3 and rare erythrocytes (not characterized), glycorrhachia 52 mg/dL, CSF protein increase 35.8 mg/dL and LDH 30 UI/L. Gram staining revealed microorganisms and antigen study was not carried out, due to insufficient sample. Microbiological and anatomopathological liquor exams are still underway.

Angio-MRI-CE and Visual Evoked Potentials were also requested.

On the 3rd day of hospitalization, while waiting for the complementary diagnostic exams, the patient reported worsening of the headache in the last 12h, at that time very intense, associated with an episode of vomiting in morning and photophobia, making orthostatism impossible. Objective examination showed neck stiffness, without any other neurological alterations. As a result, it was decided to carry out emergency CT-CE, with intravenous contrast, which revealed "prepontiac discrete subarachnoid hemorrhage of the suprasellar cistern and proximal portion of the Sylvian fissures" and by 3D reformatting, "saccular aneurysm of the right posterior communicating artery direct perpendicularly behind and with approximate dimensions of 7.5 mm at the widest anteroposterior axis".

The patient was immediately transferred to the Neurosurgery department of a leading hospital, where she was admitted.

On the 3rd day post-rupture of the aneurysm, angiogram was performed, confirming a saccular aneurysm of the right posterior communicating artery of 8x5mm, and revealing a small aneurysm of the carotid cavum on the same side. The first was embolised, without complications. On the same day, due to the reemergence of paresis in the left lower limb, grade 2/5, a new CT-CE was performed, revealing haematic density of the fourth ventricle, basal cisterns, Sylvian fissures and occipital horn of the left lateral ventricle, with an incipient hydrocephalus on the same side. A diagnostic hypothesis of repeat subarachnoid hemorrhage was assumed, either pre- or intra- intervention angiography. The angiography




Images of the CT-CE without IV contrast, on the 3rd day post-rupture of the aneurysm, soon after embolization. Areas of haematic density are highlighted, the fourth ventricle (1), basal cisterns (2), Sylvian fissures (3) and occipital horn of the left lateral ventricle (4). Incipient hydrocephaly is also visible (5).

**FIG. 4**

was repeated, which did not show a new aneurysmal rupture or thromboembolic occlusion, and did not suggest cerebral vasospasm.

On the 3rd day post-embolization, with progressive improvement of the headache and monoparesis (grade 4/5), transcranial Doppler was performed, and it was concluded that there was a moderate degree of diffuse arterial vasospasm, therefore the basic therapeutic measures for antivasospasm were reinforced.

On the 4th day post-embolization, she presented only a slight decrease in muscle strength when extending the left leg and foot.

On the 7th day, the CT-CE was repeated, with no evidence of haematic densities or hydrocephaly.

The patient began physiotherapy for motor rehabilitation on the 9th day post-embolization.

On the 16th day, without headaches, although still with some degree of paresis of the third right cranial nerve, as well as paresis on dorsiflexion of the left foot, she was clinically stable, and was referred for consultation with Neurosurgery and Physiatrics.

## DISCUSSION

In this case of a 40-year-old woman, apparently with a history of around one year of progression of neurological deficits in different locations of the CNS, a diagnostic hypothesis of demyelinating disease, in particular, Multiple Sclerosis, must be considered.

A systemic auto-immune disease, with involvement of the SNC, is another hypothesis to consider.

On the other hand, bearing in mind the patients' context, which is favorable for sexually transmitted diseases, one cannot exclude *a priori* the possibility that the neurological alterations may be secondary to SNC infection by the spirochete *Treponema pallidum* – Neurosyphilis – or by the Acquired Immunodeficiency Virus. However, the presence of negative serologies excluded these hypotheses. Furthermore, the evolution of the neurological symptoms in the case of our patient, being very prolonged, made a hypothesis of neuritis/viral encephalitis unlikely. Another possible etiology to consider would be intracranial parasite infection. Admitting that the different neurological signs and symptoms do not make a single clinical condition, and remembering the presence of two possible recent foci of consecutive infection on the right, an acute otitis media and a tooth abscess, it would be legitimate to propose a hypothesis of pyogenic infection/abscess intercepting the pathway of the third cranial nerve. However, apyrexia and the non-existence of significant increase in inflammatory parameters did not favor any of these diagnostic hypotheses. Paresis of the oculomotor nerve can, equally, take the form of a post-infection viral syndrome.

Although the headaches were persistent, no abrupt onset of the headaches of great intensity was observed, therefore clinically, the presence of a subarachnoid hemorrhage seemed unlikely.

On the other hand, in the case of ischemic vascular disease (for which there is at least 1 known risk

factor – smoking) alterations in the CT-CE would be expected, due to the duration of the symptoms.

Other diagnostic hypotheses include intracranial, primary or secondary tumors, or a paraneoplastic syndrome.

If we focus only on the reason for going to the Emergency Service, "progressive worsening of the headaches, diplopia and right palpebral ptosis with 2 weeks of evolution", the diagnostic reasoning should be different from what was actually followed. After suspicion of paresis of the third cranial nerve – in which the diplopia, where present, is binocular; or the direct and consensual ipsilateral photomotor reflexes are diminished or abolished; or the ptosis, where present, is asymmetrical and does not improve after resting the eyelid; or the eye is in a permanent "downwards and outwards" position, or one or more of the following movements is impossible: adduction, upward-inward, downward-outward or upward-outward gaze, or the accommodation reflex is diminished or non-existent – it must be determined that this occurred in isolation or is associated with other alterations in the neurological exam, and whether not the pupil is affected.[1] If there is pupil involvement, it is necessary to quickly rule out intracranial aneurysm,[1] a potentially fatal situation that requires urgent intervention.[1,4] The most frequent is that of the posterior communicating artery, which presents a risk of imminent rupture within hours or days.[1,2] Therefore, angio-CT or angio-MRI should be requested, or possibly angiography.[3] If the pupil is spared, despite thinking of other more probable etiologies, such as micro-infarctions of the nerve associated with high blood pressure, diabetes mellitus or other vasculopathies, the patient should be carefully monitored.[3]

In the clinical case presented here, a diagnosis of aneurysm of the right posterior communicating artery was made, pressing the third cranial nerve on the same side, and culminating in rupture, with subarachnoid hemorrhage. However, the symptoms of ataxia with 1 year of evolution have still to be explained, as well as the acute confusional syndrome two months previously, for which reasons the patient remained as an outpatient of Internal medicine, for monitoring and complementary diagnostic exams that were not carried out, or were previously negative, seeking to rule out demyelinating disease or system autoimmune pathology with involvement of the SNC. ■

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