

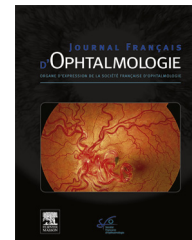


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ORIGINAL ARTICLE

# Mydriasis revealing vascular and osteodural compression of the oculomotor nerve: An observational study on five cases



*Mydriase révélant une compression vasculaire et ostéodurale du nerf oculomoteur : à propos de cinq cas*

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## KEYWORDS

Oculomotor nerve injuries;  
Mydriasis/etiology;  
Tonic pupil;  
Nerve compression syndromes

## Summary

**Purpose.** – To describe a form of neurovascular compression of the third cranial nerve (CNIII) in idiopathic mydriasis, in which a neurovascular “conflict” exists between the oculomotor nerve, the posterior communicating artery and the clinoid process, using high-resolution magnetic resonance imaging (MRI) with fast imaging employing steady acquisition (FIESTA) sequences.

**Methods.** – An 18-month prospective, observational and monocentric case series report was performed. MRI was performed on 5 consecutive patients with idiopathic, unilateral, persistent and nonreactive mydriasis (pure intrinsic palsy of the CNIII). Patients with diplopia, ptosis or ophthalmoplegia were excluded. Cerebral MRI focused on the CNIII pathway from the mesencephalon to the cavernous sinus entry, particularly on the cisternal segment: image acquisition was performed on a 3 Tesla MRI; the protocol included fast imaging employing steady acquisition (FIESTA) and three-dimension time of flight (3D TOF) sequences.

**Results.** – All patients presented a neurovascular compression point, involving the CNIII clamped between a tortuous posterior communicating artery (PCoA) and the posterior clinoid

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process at the entrance of the cavernous sinus. No cases occurred in the root entry zone. There was no compression on the contralateral side. No tumors or aneurysms were found. Thus, the mydriasis was caused by CNIII compression.

**Conclusion.** – MRI, including FIESTA sequences, revealed a new type of neurovascular conflict between the CNIII, PCoA and posterior clinoid process in patients with incomplete oculomotor palsy. Non-aneurysmal CNIII compression should be considered as a differential diagnosis in the work-up of idiopathic mydriasis. The role of MRI in the work-up of anisocoria should be considered.

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## MOTS CLÉS

Blessures du nerf oculomoteur ;  
Mydriase ;  
Étiologie ;  
Pupille tonique ;  
Syndromes de compression nerveuse

## Résumé

**Introduction.** – Le but de ce travail est de décrire un conflit neurovasculaire entre le nerf oculomoteur, l'artère communicante postérieure et le processus clinéoïde révélé par une mydriase unilatérale et diagnostiqué par une IRM en séquence *fast imaging employing steady acquisition* (FIESTA).

**Matériel et méthodes.** – Il s'agit d'une étude prospective, observationnelle et monocentrique incluant sur 18 mois des patients consécutifs présentant une mydriase unilatérale, persistante et isolée (paralysie intrinsèque pure du nerf oculomoteur) sans qu'aucune pathologie n'ait été mise en évidence par un examen standard clinique et scanner cérébral. Étaient exclus les patients présentant un signe de paralysie extrinsèque du nerf oculomoteur (diplopie, ophtalmoplégie, ptosis). Une IRM a été réalisée incluant des séquences FIESTA et *three-dimension time of flight* (3D TOF).

**Résultats.** – L'IRM a révélé chez ces patients un conflit entre le nerf oculomoteur, l'artère cérébrale communicante postérieure et le processus clinéoïde à l'entrée du canal ostéodural. Ce conflit est localisé à distance de la *root entry zone*. Il n'y pas de conflit controlatéral. On ne retrouve ni processus tumoral, ni anévrisme vasculaire.

**Conclusion.** – Ce type de conflit non anévrysmal doit désormais être évoqué face à une mydriase isolée. L'apport de l'IRM dans le bilan étiologique des anisocories est à considérer.

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## Introduction

Chronic pupillary impairment can remain unknown by the patient. The demonstration of an isolated and dilated non-reactive pupil in an awake and alert patient should primarily evoke the diagnosis of Adie's pupil [1,2] or pharmacological blockade [2]. Other conditions should be considered, including an iris trauma [3,4] or iris post-inflammatory synechia. Third cranial nerve (CNIII) paresis should rarely be considered [5], as pupillary changes would then occur along with ocular motility abnormalities. However, despite extensive and complete evaluations, some cases of mydriasis remain unexplained.

Neurologic clinical syndromes may be caused by non-aneurysmal compression by adjacent vessels against the cranial nerve root entry zones (neurovascular conflict). Janetta provided the first description of a neurovascular conflict, responsible for hemifacial spasm [6,7]. Other clinical conditions could be caused by vascular compression of cranial nerves, mainly strabismus, hemifacial spasm, superior oblique myokymia and trigeminal neuralgia [7–10]. However, neurovascular compression has never been considered as a cause of mydriasis in clinical differential diagnoses.

In the present study, we evaluate the potential role of magnetic resonance imaging (MRI) in evaluating idiopathic mydriasis, using the fast imaging employing steady state acquisition (FIESTA) in balanced gradient echo, which allows for the analysis of the CNIII pathway and its relationship to the posterior communicating artery (PCoA) in the cisternal segment.

## Patients and methods

It is a prospective, observational case report series, of patients presenting common ophthalmic features, in particular the pupillary status. Five consecutive patients were included in the series, during 18 months, 3 males and 2 females (20–41 years, mean age 33.8 years). They all presented a unilateral, persistent and nonreactive mydriasis, three from the right and two from the left, lasting on mean of 2.5 months (15 days–4 months). Old photographs were examined, finding no mydriasis. In four patients, mydriasis was discovered incidentally. In one patient, mydriasis was discovered with photophobia. Each patient underwent emergency imaging at the time of anisocoria discovery,

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