



# Ischemic chiasmal syndrome associated with posterior communicating artery (PCoA) and tuberothalamic artery (TA) infarction: a case report

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## Abstract

Lesions affecting the body of the optic chiasm typically produce bitemporal hemianopia. The blood supply comes from the anterior communicating artery, anterior cerebral, posterior communicating, posterior cerebral, and basilar arteries. We herein report a young patient admitted to the emergency department with acute confusion, left-sided hemiparesis, hemihypoesthesia, and dysarthria. Bitemporal hemianopia was detected after resolution of confusion. On cranial magnetic resonance imaging (MRI), infarction in the right anterolateral thalamus in the territory of tuberothalamic artery (TA) and in posterior chiasma in the territory of the posterior communicating artery (PCoA) was revealed. Cerebral MR angiography showed luminal irregularity of the PCoA. The patient was presented to draw attention to the rare entity ischemic chiasmal syndrome.

**Keywords** Ischemic chiasmal syndrome · Posterior communicating artery · Tuberothalamic artery

## Introduction

The optic chiasm lies above the sphenoid bone, over the diaphragma sellae. Chiasmal disorders have been classified as intrinsic and extrinsic diseases: intrinsic causes including congenital malformations, trauma, chiasmal neuritis, and tumors arising within the chiasm and extrinsic etiologies resulting from mechanical compression due to sellar, parasellar, and cavernous sinus lesions which are more common. Pituitary tumors are known to be the most common cause of chiasmal syndrome in adults [1, 2].

Due to anatomical variations, chiasmal disorders can present with different visual symptoms and signs. Bitemporal

hemianopia is the classic finding of lesions affecting the body of the chiasm. The whole hemifield, upper or lower quadrants, can be affected. However, both due to individual variations and growth pattern of the compressive lesions, defects mainly involving the visual field of one eye can be seen when one optic nerve is more severely affected. Homonymous hemianopia can be detected in case of extension to the optic tractus [2]. Affection of the macular axons results in the loss of vision. Optic disc pallor indicates a more chronic process [1, 2].

The blood supply is variable as well. Branches coming from the anterior communicating artery (ACoA) and anterior cerebral artery (ACA) supply the superior part, sparing the

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central chiasm. Branches from the internal carotid artery, posterior cerebral artery, and the posterior communicating artery (PCoA) supply the inferior part. The central chiasm drives an arterial blood supply from the inferior group of vessels [3, 4]. Chiasmal infarction has been reported to be very rare due to wide collateral supply [1]. However, in another study, anastomoses have been reported to be absent in the extrinsic or intrinsic vascular network with wide interindividual variations [5].

We herein report a young patient with chiasmal infarction within the territory of the PCoA associated with a tuberothalamic artery (TA) infarction.

## Case report

A 35-year-old male was admitted to the emergency department with acute confusion, left-sided hemiparesis, and dysarthria. The neurologic examination performed a few days later after resolution of confusion revealed bitemporal hemianopia (Fig. 1a) and left-sided hemihypoesthesia. Visual acuity was 20/20 in both eyes. Extraocular

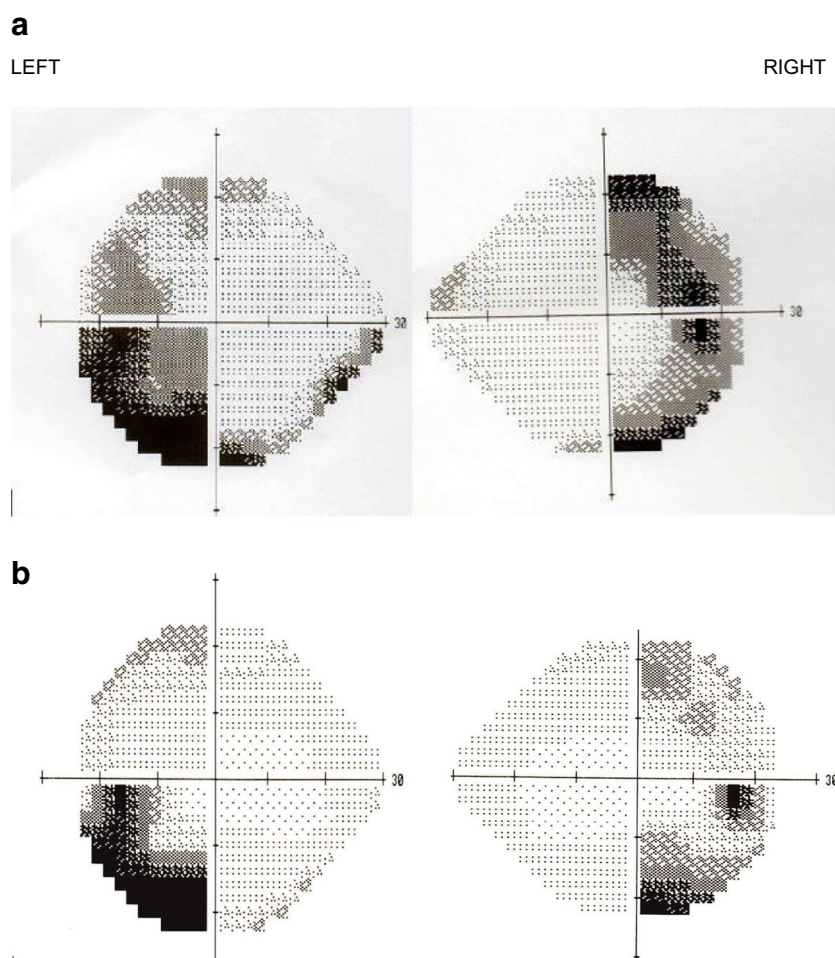
movements and pupillary responses were intact. Optic discs were normal bilaterally. He had hypertension (HT), hyperlipidemia (HL), and history of cigarette smoking for 10 package years.

Physical, cardiovascular, and respiratory system examinations were unremarkable. There were no lymphadenopathies, skin rash, and oral or genital ulcers. Pathergy test was negative. Body mass index was 27.6 (normal range 25–29.9).

Hemogram, fasting blood glucose, HbA1C, liver and kidney function tests, thyroid function tests, and vitamin B12, folic acid, ferritin levels were within normal limits. *Peripheral blood smear was normal.* Fasting blood triglyceride was 726 mg/dl (0–150), VLDL was 145 mg/dl (10–40), and cholesterol was 206 mg/dl (0–200).

Coagulation parameters (protein C, protein S, prothrombin time, international normalized ratio, activated partial thromboplastin time, D-dimer, fibrinogen), vasculitis markers, infectious disease markers (Epstein-Barr virus, cytomegalovirus, herpes simplex virus, varicella-zoster virus, toxoplasmosis, rubella, HIV and *Toxocara* serology) did not reveal any abnormality.

**Fig. 1** (a) Visual field defects on admission. (b) Visual field defects recorded 6 months later



*Neuromyelitis optica antibody* IgG and *anti-myelin oligodendrocyte glycoprotein* were negative. The serum angiotensin-converting enzyme level was 13 U/L (8–53).

Hormonal data showed normal blood concentrations of adrenocorticotrophic hormone, cortisol, *luteinizing hormone*, follicle-stimulating hormone, testosterone 1.4 *alfa androstenedion*, and sex hormone *binding globulin*.

An abnormality on behalf of cardioembolism could not be detected on electrocardiography (heart rate, 69/min), *Holter electrocardiogram*, or *transthoracic echocardiography*. Thoracic computed tomography with contrast was normal.

Cranial magnetic resonance imaging (MRI) revealed hyperintense signals in the right anterolateral thalamus in the territory of TA and in posterior optic chiasm on diffusion-weighted images and matching hypointense signals on apparent *diffusion* coefficient sequence. On fluid attenuation inversion recovery (FLAIR) and *T2-weighted images*, the same regions were hyperintense. Cerebral MR angiography showed luminal irregularity of the PCoA (Fig. 2).

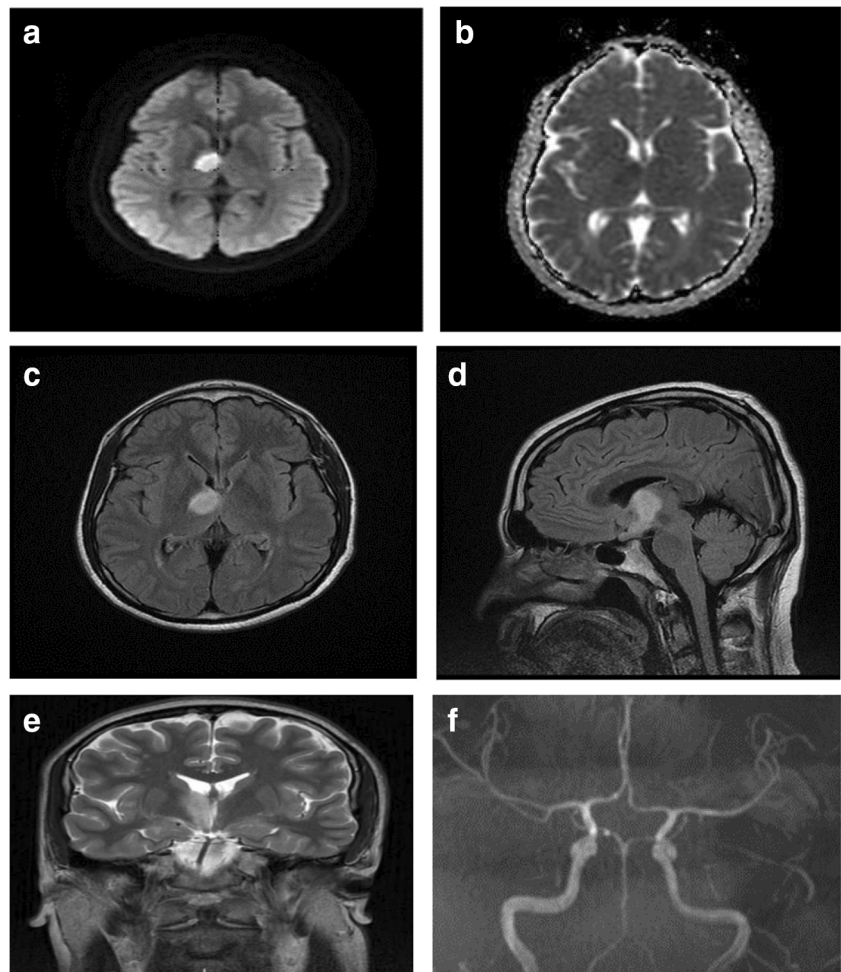
A follow-up visual field performed 6 months after the insult showed prominent regression of the bitemporal field defect (Fig. 1b).

## Discussion

PCoA arises from the internal carotid artery and provides blood supply to the inferior and central optic chiasm [3–5]. TA is a branch of the PCoA. TA supplies the intralaminar nucleus, ventral internal medullary lamina, and mammillothalamic tract in the paramedian thalamic area [6].

In the light of anamnesis, neurological examination, laboratory tests, and cranial MRI, our patient was diagnosed as stroke involving the PCoA. Confusion, left-sided hemiparesis, and hemihypoesthesia were due to infarction within the territory of TA, whereas bitemporal hemianopia was secondary to infarction involving the central part of the optic chiasm [3–5]. As described in a previous report [5], individual variations with absent collateral supply seem to be the mechanism

**Fig. 2** (a) Diffusion-weighted magnetic resonance imaging reveals right anterior thalamic hyperintense signals. (b) Apparent diffusion coefficient magnetic resonance imaging reveals right anterior thalamic hypointense signals. (c) Axial FLAIR magnetic resonance imaging reveals right anterior thalamic hyperintense signals. (d) Sagittal FLAIR magnetic resonance imaging reveals thalamic and chiasmal hyperintense signals. (e) Coronal T2 magnetic resonance imaging reveals chiasmal hyperintense signals. (f) Cerebral MR angiography showing luminal irregularity of the PCoA



underlying the occurrence of the rare chiasmal ischemic syndrome in this patient.

As he was 35 years old, all young stroke etiologies were searched but nothing could be detected other than HT, HL, and smoking. An increase in ischemic stroke in younger adults has been reported in recent studies in addition to an increase in traditional stroke risk factors that are typically common among older adults like HT, dyslipidemia, diabetes mellitus, tobacco use, and obesity [7–9]. Atherosclerotic stroke, due to the presence of multiple modifiable risk factors, was our diagnosis in this patient. Associated hypopituitarism was excluded by extensive laboratory tests for hormones.

In a previous report, two cases studied with four-vessel angiography tortuosity of the large cerebral vessel and slight atherosclerotic changes have been reported [10]. In our patient, MR angiography was performed revealing just luminal irregularity of the PCoA. An improvement in the visual field defect was detected on the sixth month follow-up as reported in the previous study [10].

Our patient was confused at the time of admission to the emergency department, and bitemporal hemianopia could be detected a couple of days later after resolution of confusion, indicating the importance of follow-up examinations in patients with altered consciousness.

This patient was presented to draw attention to the rare ischemic chiasmal syndrome.

### Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

**Ethical approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the

institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Informed consent** Informed consent was obtained from the individual participant included in the case report.

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